



# OFFICE GASTROENTEROLOGY

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## Preface

Such a vast amount of material relating to research in gastroenterology and nutrition is being produced that even a specialist can find time to read only a small percentage of the published articles. As a result abstract journals and abstract departments of all kinds of publications are constantly growing. Some abstracts are accurate, many are deceptive. Newspapers and even books based at least in part on these abstracts abound in references to them. Meanwhile the public is being deluged with pseudo scientific medical news by science writers in magazines and newspapers and by commentators and advertisers on the radio and television. Doctors often hear of new medical discoveries first from their patients. Many practitioners are depending on the information they obtain from the publications and detail men of pharmaceutical houses. Many of the new pharmaceutical preparations often not sufficiently tested experimentally to insure their safety will in themselves cause distressing symptoms for which further medications are prescribed. The result is that patients today are receiving more different kinds of medication often purely for the relief of symptoms than did the patients of the old ridiculed shot gun era. Frequently all that these patients require in order to make a prompt recovery is complete withdrawal of all medication.

For many years I was waiting for the publication of a practical monograph which would present simple and specific information regarding gastrointestinal diseases and their treatment. I even had thought of writing such a book. When my friend Dr. Henry L. Bockus told me that he was writing a book I knew it would be all that I had hoped for and I gave up the idea of writing one myself. The Bockus book proved to be a monumental encyclopedic three volume production the greatest book on gastroenterology ever written. The advances made in the field of gastroenterology since its publication have given me the feeling that the time has now come when a smaller book, a monograph based on personal experience would serve a useful purpose. The result is this volume. After forty years of experience in teaching gastroenterology to students in internes and physicians in the Long Island College Hospital (now State University of New York College of Medicine at New York City) and in



postgraduate courses in various parts of the country and having written a couple of hundred papers published in a variety of medical journals I feel that writing of my experiences may prove of value. Except for a few instances in which I have had to rely on the experience of others what I have written is based on personal experience in the care of patients. Although in the past I theorized regarding the proper treatment for diseases of the gastrointestinal tract I have never written anything which has not stood the test of time in my own practice. This is true of what appears in this book. I have always tried to present my material based originally on physiological concepts applied to diagnosis and treatment in such a way as to encourage the reader to think for himself to give no treatment without good reason.

I have tried to arrange the material in this book in logical sequence. First an introductory chapter covers the general concepts of gastrointestinal physiology, pathology and treatments including a simplified discussion of dietary indications. The reading of this chapter is essential to an understanding of the subsequent parts of the book. There follows a section on diseases which may affect any part of or the whole gastrointestinal tract including a chapter on intestinal parasites. The next section covers briefly the diseases of each individual part of the gastrointestinal tract from a chapter on the esophagus to one describing the medical aspects of diseases of the anus and rectum. The next section discusses diseases originally affecting other parts of the body which may produce gastrointestinal symptoms or actual lesions. Under each disease physiologic indications for treatment are discussed and treatment including diet and medication is specifically outlined. When the treatment should be similar to one previously prescribed for another disease this is mentioned and the page on which it is described is given. This arrangement not only saves time in looking up the treatment in the index but also saves time and space which otherwise would be lost by duplication. The book abounds in these references.

After due consideration and with the approval of the publishers this book does not give space to a bibliography. We felt that the usual long list of references in books are probably never used by over 90 per cent of readers especially the busy practitioners for whom this book has been written. I recently studied practically all the current and many of the older books covering phases of gastroenterology and endeavored to make use of all new material from the literature. I have modified what I read in the light of my own experience. I apologize to the many authors for not mentioning their names.

In order to save space illustrations have been kept to a minimum. Permission has been obtained and credit given for illustrations from various sources. All but a few of the x-ray pictures used to demonstrate typical lesions were made by my friend Dr. A. L. I. Bell. Nearly all are

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based on studies he made in the crises of my patients over the past forty years during which we have worked out such problems together Dr Herbert Friedman and Dr Lewis L Immerman have permitted me to use some of their films I am greatly indebted to the outstanding photographic expert Mr Stephen Montes for the fine prints he made for the x ray illustrations He has made well over a thousand prints and lantern slides for me in the past

I feel that I must give credit to my colleagues both in my own department and in the other departments of the Long Island College Hospital for their cooperation and inspiration over many years I also express my appreciation to the many practitioners who have given me the opportunity to study and treat their patients

This is my first book I never realized how much work is entailed in writing and publishing a medical book I have been astounded at and deeply appreciative of the tremendous amount of help and encouragement given me by the Saunders organization I wish to express my appreciation and thanks to Mrs Natalie J Hoyt for editing and arranging the material and for preparing the fine index

If any credit is due for the writing and preparation of this book it must be given entirely to my secretary Mrs Estelle Wulsen who originally inspired me to write it with whom I discussed every detail of what to write and who typed and retyped the original manuscript Her constant encouragement and cheerful cooperation helped to keep me at work when I showed any tendency to lag

ALBERT F R ANDRESEN

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## SECTION I

### Introduction



# A Plan of Attack on Gastrointestinal Diseases

Various studies and estimates have demonstrated that at least 20 to 25 per cent of all patients consulting physicians or applying at clinics and hospitals both civilian and military complain principally of symptoms referable to the gastrointestinal tract. Unfortunately, most medical school curriculums devote too little time to gastroenterologic subjects. The usual internships and residencies, even in gastroenterology, while giving the candidate excellent experience in the more serious lesions requiring hospitalization, fail to familiarize him with the much more frequent conditions which must be handled on an ambulatory or home care basis. A considerable number of books on gastroenterology have been produced, some too long and detailed for practical use by the general clinician, others devoted only to certain phases of the subject as viewed by the author or a group of authors. Medical journals of all types are filled with scientific articles and advertisements about the diagnosis and treatment of gastrointestinal conditions. Both books and journals devote much space to references to the literature which have often been inaccurately interpreted by the authors and besides are rarely consulted by the readers.

In recent years many articles have been devoted to reports on the action of new drugs, the work subsidized by the manufacturers. News papers, magazines, programs, billboards and other advertising media give advice to the sufferer from gastrointestinal symptoms. It is difficult today for a physician to keep up to date on medical matters without reading several of the lay magazines which publish articles on old and new phases of medical practice written by laymen as well as by pharmacists, nurses, dentists and physicians. It is small wonder that surgeons and other specialists are consulted by a steady stream of patients who have been treated empirically or symptomatically with neglect of the underlying causes of symptoms. The mounting death rate from gastrointestinal cancer and the persistently high death rate from acute abdominal calamities, notably appendicitis, can be attributed to neglect caused by confusion from uncritical reading of all these media.

*Before treating* any patient with gastrointestinal symptoms, whether acute or chronic, the clinician should give adequate time to a study to determine the cause of the symptoms. Although it is true that many acute conditions will subside spontaneously and will appear to improve

after any medication or placebo many others may go on to a fatal termination or may be followed by years of suffering and disability due to neglect in making a diagnosis. A doctor who does not have time to study a patient should not treat a gastroenterologic case.

In studying the gastrointestinal patient the clinician should be cognizant of certain essential facts:

- 1 He should have an understanding of normal *gastrointestinal physiology*.

- 2 He should know how disease affects physiologic processes which will aid in the interpretation of *symptoms*.

- 3 He should use a systematic outline in obtaining the *history* so as not to miss important points.

- 4 He should make a complete *physical examination* so as not to overlook what may appear to be minor defects.

- 5 He should use the necessary special *instruments* to determine if possible the exact lesions responsible for the symptoms.

- 6 He should make use of such *laboratory tests* as may be indicated.

This sounds like a formidable program but if carefully carried out it will be worth the time expended. Instrumental and laboratory methods of study were put last in the list because they should be regarded as adjuvants rather than as the principal methods of diagnosis. If their findings disagree with the clinical opinion regarding the diagnosis careful rechecking should be done before the carefully considered clinical diagnosis is abandoned. Each of these six subjects will be discussed in the order named.

## Gastrointestinal Physiology

For practical purposes the gastrointestinal tract and its appendages the biliary tract and pancreas may be considered one organ the function of which is to process ingested material to permit its absorption and to get rid of whatever is not used in the process in addition to waste materials such as excretions, bacteria and deciduous mucosa. In single celled organisms absorption takes place through the surrounding membrane in some cases aided by pseudopods in others by secretion from the surface. In man the inversion and diversification of these external membranes through evolution produced the gastrointestinal tract so that it can be truly considered that any material lying in the lumen of the tract is really outside the body.

The processing thus really taking place on the outside consists in digestion of foodstuffs by the alkaline saliva (starches) the acid gastric juice (proteins) and the alkaline duodenal and pancreatic juices and bile in the upper part of the tract. These *digestive secretions* of the gastrointestinal tract consist of enzymes admixed with alkali or acid according to the requirements of each enzyme. Mucus is an important

secretion because it both promotes lubrication and protects the mucosa from injury by chemical or mechanical irritants. Mucus is poured out freely in the presence of any irritation. Each secretion is controlled directly by the presence of food indirectly by various nervous influences and is itself influenced by disease. The normal gastrointestinal tract and its appendages produce adequate secretions to digest all kinds of food stuffs required by the body metabolism. The products of the digestion of proteins, fats and carbohydrates are in a form easily absorbable through the mucosa.

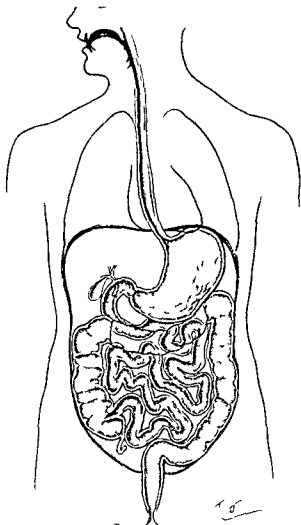


Figure 1 The normal alimentary tract (Small bowel has been shortened for convenience of illustration) (Courtesy of Wyeth Laboratories)

*Absorption* into the body fluids takes place (1) a little in the stomach (2) mostly in the small intestine the membrane of which is thrown into folds (villi) to increase the absorptive surface and (3) a little mostly water and a few salts in the colon. In order to maintain adequate nutrition it is necessary to eat adequate quantities of each of the foodstuffs mentioned and also of certain accessory food factors the vitamins and minerals. The requirements of each will be discussed under Physiologic Treatment (p. 20).

### Peristalsis

Progress of the material through the gastrointestinal tube with delays for storage permitting the processing and absorption is accomplished by the muscle layers of the gastrointestinal wall which alternate between a state of tonus and one of relaxation thus keeping a firm hold on the contents. The muscularis mucosae by means of peristaltic waves moves the contents downward.

Although its authenticity has been questioned Alvarez's theory of gradients in the gastrointestinal tract best explains how peristalsis is accomplished and how it is interfered with by disease. Alvarez demonstrated that material placed in the lumen of any part of the gastrointestinal tract progresses downward in a caudad direction. Successive segments of the wall of the tract are progressively less active and irritable show less metabolism and less forceful and frequent contractions and in every way demonstrate a weaker response to any stimuli. The content therefore can move only from the stronger to the weaker area. He showed that this would take place even when all nerve supply was cut off from the wall. With the nerves intact he demonstrated the influence of nervous stimuli which might at times nullify or enhance the effects of the gradient slowing or accelerating the peristalsis. Normally the steepness of the gradient is modified by the sphincters at the cardia pylorus ileocecal junction Cannon's ring (at the junction of the first and middle thirds of the transverse colon) and at the rectum. These sphincters points of increased irritability act as "bumps" in the gradient producing an effect such as that caused by a moderately elevated area in the side of a hill down which a stream is flowing. Above each bump there occur some reverse waves in the intestinal wall until the increasing bulk of their contents and their acceleration (raising the gradient) force them over the "bump." A tendency to greater force and speed follows for a short time after they have passed the temporary obstruction.

Similar effects are produced when some part of the gastrointestinal tube is narrowed by disease or pressure or when its irritability is increased by inflammation or other source of irritation. Increased irritability and delay at one point may be the result of (1) a spastic sphincter

or (2) an area of inflammation as in peptic ulcer localized enteritis appendicitis sigmoiditis or proctitis or (3) an area of actual narrowing from any of the diseases mentioned from intrinsic neoplasm or external pressures such as enlarged prostate uterus adnexa or other abdominal or pelvic tumors or (4) contact with an inflamed or otherwise diseased organ such as gallbladder pancreas or genitourinary tract. The effect of the reverse waves originating at such an area was called *retrostalsis* by Alvarez who was careful to point out that by this is not meant reverse peristalsis with actual carrying of contents to a higher level but only traces going upward against the gradient with no actual movement of contents. These waves when sufficiently forceful upon reaching the next higher sphincter an irritable area will produce a spasm there and the process is again repeated.

Such disturbed peristalsis causes in a patient a feeling of so-called peristaltic unrest "gas" "butterflies" in the stomach or simple "indigestion." If the pyloric sphincter is made sufficiently spastic it may cause gastric content to regurgitate into the lower esophagus producing heart burn to the upper esophagus causing a feeling of "gas" and encouraging air swallowing and belching into the pharynx producing eructation or into the mouth producing regurgitation or actual vomiting. This whole sequence of events may occur in the course of any abdominal disease affecting any part of the gastrointestinal tract even to the point at which vomiting occurs with anal sphincter spasm resulting from an anal fissure. These symptoms collectively have been called *retrostaltic symptoms* and will be so called in this book rather than the term "reflex symptoms" frequently used in the past without definite explanation. Retrostaltic symptoms usually occur soon after ingestion of food. They are then principally due to pylorospasm. They may occur at the time of greatest irritability at some point lower down when that area is most active that is when contents are reaching it and being delayed. Retrostaltic symptoms also result from nervous impulses. They are therefore only of significance as indicating that some cause of gastrointestinal irritation is present somewhere within or without the gastrointestinal tract.

The increased peristalsis beyond the "bump" in the gradient is not so frequently noted. The initial small intestinal hypermotility noted in the x-ray study in cases of peptic ulcer is an example as is the diarrhea of regional enteritis or an occasional case of appendicitis and of ulcerative colitis diverticulitis and colonic neoplasms. Under each disease the effect on motility will be discussed in detail.

*Rate of progress* of food through the gastrointestinal canal can best be followed by x-ray pictures taken at intervals after a barium meal. Normally the mixture passes rapidly down the esophagus with slight delay in the vestibule above the sphincter at the cardia. The stomach begins to



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scribed by patients as gas and may be a distressing symptom. Aero-phagia or air swallowing is the principal cause of belching and frequently of abdominal distention and flatus. It may be purely a bad habit starting in childhood from imitating in air swallowing parent or employee. Usually it is due to efforts at bringing up "gas" which the patient assumes to be the cause of some kind of abdominal or thoracic pain or distress.

The mechanism consists in closing the lips tightly and swallowing the air in the mouth. The air may not actually enter the esophagus but on reaching the pharynx may be forcibly and loudly expelled. This process may be continuous causing exhaustion of the patient and annoyance to those around him. On the other hand the air may pass down the esophagus and if rapidly swallowed may cause enough distention of the lower end to produce considerable substernal pain. The patient is happy when the air comes up and relieves the pain. In other persons the air goes into the stomach and when not in large amount as with air swallowed during a meal may cause no distress being seen on x-ray picture as the "gas bubble" or "magenblase." Some patients will distend the stomach with swallowed air causing much "bloating" and discomfort and at intervals a loud belch as the accumulated air comes up. Again there is relief. In extreme cases I have observed under the fluoroscope



Figure 2 Ileus from aerophagia (1) Air in stomach (2) air in small intestine (3) air throughout colon

empty liquids at once and an ordinary meal should be completely liquefied and evacuated in six hours with the head of the meal approximately at Cannon's ring. In twenty four hours the colon is filled to the recto sigmoid junction. At this stage the contents still in the cecum and ascending colon are liquid; those in the descending colon are dehydrated.

Between twenty four and forty eight hours most or at times all of the nonabsorbable content which has accumulated in the descending colon and sigmoid has been expelled. It can be seen that contents are dehydrated as a result of reverse and forward peristalsis between the cecum and Cannon's ring, the only place where real reverse peristalsis is normal. By means of peristaltic rushes originating in the cecum usually induced by peristaltic waves originating in the stomach as a result of ingestion of food, the partly dehydrated contents are carried to the rectosigmoid junction where they are stored for twenty four to forty eight hours according to their bulk and further dehydrated.

Finally, a particular powerful peristaltic rush will carry the content (the feces) into the rectum producing the normal desire for *defecation*. The process of expelling the rectal contents is accomplished partly by involuntary peristalsis partly by voluntary assistance of the various muscles acting on the lower rectum and partly by relaxation of the sphincter. The frequency and size of bowel movements are determined by the amount of undigestible residue present in addition to secretions of the upper intestines, liver and pancreas, desquamated epithelia and other debris and millions of bacteria, dead and viable, the amount of water to keep it soft and by habits of regularity. On a high residue diet with plenty of water a normal person may have up to three formed, often soft stools a day; on moderate attention to diet one formed stool a day; and on a low residue diet with insufficient fluids there may be defecation only once or twice a week. Mere constipation is usually due to bad habits, not to disease.

## Symptomatology

Gastrointestinal symptoms are an expression of disturbed physiology caused by disease. The most frequent and important symptoms and the parts of the gastrointestinal tract with which they are usually associated are as follows:

### Retrostaltic Symptoms

Retrostaltic symptoms occur as a result of some intrinsic or extrinsic source of irritation affecting any part of the gastrointestinal tract and have no specific significance. They were discussed under Gastrointestinal Physiology (p. 7).

*Belching* Belching included among retrostaltic symptoms is de

or rectum. This pain is usually severe and sharp. It may occur with any disease causing marked retrostaltic symptoms and is most severe in ulcers close to sphincters as in the lower esophagus, pyloric region and in the rectum. (3) The pain due to *overdistention* of the wall with stretching of the peritoneal coat which is sensitive to pain. This is observed with ileus from any cause and occasionally from overeating. Rarely any of these three types of pain may be neurogenic in origin and may be the result of circulatory, hormone, endocrine or electrolyte imbalance. They definitely call for a complete gastrointestinal study. (4) The pain due to tearing or bursting of the peritoneal coat called *perforation* whether due to disease or injury. This causes the most intense agonizing pain.

**LOCALIZATION.** Pain may be localized over the organ involved as in the esophagus, stomach, ileum, appendix, sigmoid or rectum but perhaps even more frequently it is referred elsewhere. Pain due to diseases in the *stomach* and *duodenum* is usually referred to the mid-epigastrium but may be located only in the back in the mid or lower dorsal region or may be referred to some other area as to the right or left lower quadrant. In peptic ulcer however it will usually retain its relation to food intake (hunger pain). *Small intestinal lesions* usually cause pain in the umbilical region but it may be only in the back or may be beltlike. Being due to exaggerated peristalsis from partial or complete obstruction such pains are usually intermittent or cramplike. *Colonic pains* are usually felt below the umbilicus although they may occur in the back or on either side. They are usually cramplike although not as agonizing as small intestinal colic. *Appendiceal* and at times also *terminal ileal* pain may be over the ileocecal region although in acute lesions it may start in the mid abdomen or epigastrium and later be localized. When the appendix is retrocecal the pain may be entirely in the back or in the right upper abdomen. *Rectal* pains may be in the rectum or the sacral or suprapubic region. *Gallbladder* pains may start in the epigastrium move to the gallbladder region and then be referred around the right side to the back and to the scapula. Rarely the pains of gallbladder disease will be referred entirely to the left side even though there is no transposition of organs. *Liver* pains are rarely severe more frequently there are no pains in liver disease. If they occur as in acute hepatitis or abscess they are usually over the liver or in the back. Pains on top of the *shoulder* are usually due to peritoneal irritation high up.

**TIME RELATIONSHIP.** The relationship of pain to gastrointestinal functions will often be of great help in determining its origin.

*Pain during swallowing* especially with dysphagia places the disease in the esophagus.

*Pain immediately after eating* and often relieved by vomiting usually indicates a retrostaltic origin although it may occur at times when after

air being swallowed passing through the stomach and pylorus and dis tending first the small and then the large intestine

Belching may also be caused by gases other than air The rarest cause is fermentation of gastric contents due to stagnation from pyloric duodenal or hourglass stenosis This produces at most occasional belching of "fumes" which may smell like yeast in stenosis due to ulcer or may have a putrid odor in the presence of ulcerating cancer

Belching may also be induced by the swallowing of charged waters or wines the gas being then carbon dioxide The same gas is produced by swallowing of sodium bicarbonate other alkalies or even large amounts of saliva from chewing gum when there is free hydrochloric acid in the stomach When the air or gas comes up from the stomach it will have some of the odor or taste of the stomach contents usually of food or acid the latter called "sour eructations" The sour taste may occur even in the absence of free hydrochloric acid in the stomach being probably due to lactic or butyric acids

Patients make a big fuss over these symptoms of "gas" but can be quickly cured of air swallowing if the mechanism is explained to them They should be told that the distress which makes them want to belch is usually due to a spasm somewhere not to gas The patient should be instructed that when the feeling occurs he should open his mouth widely and breathe through it for a few minutes until the distress subsides spontaneously Enlisting the help of those near him to call attention to his compressing the lips preparatory to belching a job the wife of an air swallower usually enjoys is a great help Most patients can be cured of the habit within a week or two No medication is required

**Dysphagia** Dysphagia is a term which includes not only difficult or painful swallowing but also actual inability to push foods through the esophagus into the stomach It is usually accompanied by regurgitation of the swallowed material It may be due to organic disease of the esophagus to external pressure upon it or to spasm The patient can usually judge with remarkable accuracy where the point of obstruction is located

**Pain** Since no sensory nerves are located in the stomach or intestine pains are usually of four types

**TYPES AND CAUSES** (1) The pain of *hyperperistalsis* due to obstruction or inflammation which is usually intermittent or cramplike It is observed in intestinal obstruction in case of a stone in the neck of the gallbladder and in ulcerative colitis enteritis and other causes of diarrhea Related to this are the excessive hunger contractions causing peptic ulcer pain relieved when the contractions are stopped by putting something into the stomach (2) The pain associated with excessive contraction or *spasm* of the muscle at some point in the gastrointestinal tube especially at the sphincters most frequently the sphincter at the cardia pylorus

or rectum. This pain is usually severe and sharp. It may occur with any disease causing marked retrostaltic symptoms and is most severe in ulcers close to sphincters as in the lower esophagus, pyloric region and in the rectum. (3) The pain due to *overdistention* of the wall with stretching of the peritoneal coat which is sensitive to pain. This is observed with ileus from any cause and occasionally from overeating. Rarely any of these three types of pain may be neurogenic in origin and may be the result of circulatory, hormonal, endocrine or electrolyte imbalance. They definitely call for a complete gastrointestinal study. (4) The pain due to tearing or bursting of the peritoneal coat called *perforation* whether due to disease or injury. This causes the most intense agonizing pain.

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overeating in overfilled gastric ulcer crater is stretching the peritoneal coat

*Pain late after eating and relieved by food* is almost pathognomonic of peptic ulcer usually a benign but occasionally a malignant ulcer

*Relationship to defecation* may be important When *aggravated* during defecation and relieved afterward it suggests a rectal lesion *Preceding* defecation it may indicate a lesion higher up Pain with prolonged *constipation* suggests intestinal obstruction or ileus With *diarrhea* it implies a colonic or rectal lesion inflammatory or malignant

*Rectal pain* with defecation may come from an anal or rectal lesion or from the prostate bladder or female genitals

*Pain with exercise* or with certain postures may be due to hernia but occurs more with spinal or neuromuscular conditions It may be a cardiac pain

*Nausea and Vomiting* These symptoms like pain may be purely psychogenic in origin or the result of hormone endocrine or electrolyte imbalance Gastrointestinal disease produces them as follows

Nausea and vomiting *immediately* or soon after eating are usually a part of the retrostaltic group of symptoms and may be due to irritation of any part of the gastrointestinal tract as mentioned before

*Late vomiting* two to four hours after eating may occur with a peptic ulcer pain relieving it temporarily It may also occur soon or a few hours after eating with partial small intestinal obstruction

*Constant or repeated vomiting* with severe pain occurs with intestinal obstruction and with peritonitis (see Acute Conditions of the Abdomen p 61)

*Delayed vomiting* consisting of food recognized as having been eaten during a period of ten or twelve hours or more previously usually coming on suddenly and unexpectedly and profuse but not very forceful indicates pyloric or duodenal obstruction It may relieve a previous feeling of distention especially when it consists of a quart or more of yeasty smelling vomitus which settles into three layers on standing

*Sudden projectile vomiting* with gastric contents shot high into the air or across the room occurs in central nervous system lesions or in less severe form in acute pancreatitis

*Anorexia* Anorexia may be associated with disease of the gastrointestinal tract It is most frequently due to one of three factors (1) infections in the mouth or respiratory tract associated with bad odors and taste (2) allergy to foods or other ingestants or to certain odors or emanations which may cause allergic reactions in the mouth such as canker sores which cause not only pain but also bad odor and taste (3) merely the bad habit of not eating sufficient food at proper intervals which may have been initiated as a result of restrictions in the diet for various reasons (4) When the patient finds food unpalatable or has a

fear that food will cause symptoms less food is eaten and the habit is formed. It may thus be entirely psychic in origin a behavior problem. I always explain that *appetite* is a pleasurable psychic phenomenon developed by the habit of eating where is *hunger* to which appetite does not necessarily bear any relation is a physical sensation due to hunger contractions in the stomach. Forcing of a balanced diet with frequent feedings will usually restore appetite in such cases usually within a week or two.

*Constipation and Diarrhea* Constipation and diarrhea are frequent symptoms and may occur in connection with any gastrointestinal condition. As they are both essentially intestinal symptoms they will be discussed at length in the chapter on the Intestines (p. 301).

*Tumors and Hernias* Tumors and hernias may be classed among symptoms when the patient complains of a "lump" or a protrusion. The "lump" may be nothing but a small sebaceous cyst or a lipoma in the skin or may be so large as to be visible through the clothing. I remember a patient who came to me complaining of a "lump in the stomach" who had a uterine fibroid as large as a fetus in a full term pregnancy. Some patients who say they have hernias point to an inguinal gland or fat in the inguinal region others do not realize that they have hernias when there is bowel in the scrotum or when there is complete herniation through an epigastric operative scar. Such a hernia may be described by the patient as "flabbiness."

## Diagnosis

The diagnosis of a gastrointestinal disease depends upon careful systematic study and interpretation of the patient's symptoms of the findings on thorough general physical examination and of the information acquired from instrumental and laboratory procedures. Unless such a study is carried out in a systematic way some important factors may be left out the diagnosis may be missed and the patient harmed. Even an experienced clinician who prides himself on history taking will be benefited by following an outline. Such an outline printed on both sides of a 5 x 7 card I have used for teaching and for my own guidance during the past forty years modifying it from time to time.

### OUTLINE FOR HISTORY AND EXAMINATION

#### 1. FAMILY HISTORY

Specific State of health or cause of death of parents brothers and sisters with age of each

General Tuberculosis—carcinoma—insanity—diabetes—paralysis—invalidism—hemophilia—alcoholism—longevity—characteristic weight



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**Habits, etc.** Time in this country—occupations—amount of exercise—air—sleep—tobacco—drugs—drinking (water alcohol sodas coffee tea)  
**Armed services** Usual diet breakfast—lunch—supper—between meals (in detail)

**Gastrointestinal** As child—acute indigestion—bilious attacks—ptomaine—jaundice—worms—piles—fistula—fissure—appendix—vomiting—gas—pains X rays *Bowels* habits—cathartics—enemas—suppositories—blood—pus—slime—pain

### 3 PRESENT HISTORY

**Initial Symptoms** Time—character—continued or intermittent—increasing or decreasing

**Pain Distress** Location—radiation—severity—character (aching boring gnawing burning bursting sharp dull cramplike pressure)—steady or intermittent—time of occurrence (relation to food intake influence of food or drink)—relief or aggravation by medicine rest food vomiting posture exercise massage local applications defecation cathartic or enema

**Fullness Belching, Nausea, Regurgitation, Vomiting** (Retrostaltic Symptoms) Time—duration—frequency—character—taste—spontaneous or induced—relation to character and time of food taken—relation to condition of bowels and to menstrual dates—effect on pain

**Appetite** Degree—*anorexia*—fear of food—voraciousness—thirst

**Dysphagia** Duration—liquids or solids—which first—steady or intermittent—increasing or decreasing—associated with pain distress or regurgitation

**Flabbiness or Hernia** Location—duration—disability—support used—efficiency—pain

**Tumor** Location—time first noted—tenderness—growth or retrogression

**Weight** Usual—gain or loss—times and places weighed

**Bowels** Regularity—number stools per day—pain (relation)—feeling after  
*Constipation* duration—kind of cathartics suppositories or enemas used—pain at stool—effect on other symptoms *Diarrhea* duration—frequency—pain—tenesmus—relationship to food—character and time  
*Stools* consistency—size—caliber—color—odor—mucus—blood—pus—worms—undigested food

**Disability** Degree—amount of work—bed—cares at home

**Previous X rays** Time taken—report—doctor or hospital

**Present condition** of all other parts of body as outlined in past history

### 4 PHYSICAL EXAMINATION

**General** Body type—nutrition—hemic component—facial expression—hair and skin—color—prominent blood vessels—paralyses—deformities—stigmata

## 2 PREVIOUS HISTORY

**Under each head** Date—duration—seriousness—complications—treatment (doctor and hospital)—sequelae

**Infections** Scarlatina—diphtheria—measles—pertussis—paralysis—typhoid—pneumonia—influenza—tuberculosis—pleurisy—rheumatism—erysipelas—malaria—"blood poisoning"—vaccine reactions

**Ear, Nose, Throat** Coughs—colds—sore throats—catarrh—hoarseness—ear pains—noises—discharges—vertigo—glands—mastoids—operations

**Teeth** Care—ulcerations—abscesses—loosening—odor—extractions (reason)—artificial—x rays—root canal fillings

**Eyes** Tire—pain—inflammations—styles—blindness—haziness—specks—blurring—glasses (where obtained—oculist or optician)

**Respiratory** Cough—pain in chest (location)—expectoration (frequency and time character yellow white bloody thick thin)—x rays

**Cardiovascular** Palpitation—pain—dyspnea—syncope—vertigo—edema—varicosities—previous study—electrocardiograms

**Urinary** Micturition (frequency day and night burning painful)—edema—vertigo—eye blurring—headache—backache **Urine** (color blood odor quantity sediment stain)—urological studies—x rays

**Skin** Temperature—dryness—discolorations—itching—rash—infection—tumors **Condition of hair and nails**

**Nervous System** Fatigue—convulsions—spasms—numbness (local or general)—fainting—pains (head spine body extremities)—effect of posture—hot or cold feelings—hours of sleep

**Psychological** Stress—worry—annoyances—anxiety—sexual aberrations—memory—disposition

**Extremities** Pains—swellings—varicosities—infections—ulcers—loss of power or sensation

**Gynecological** *Menstrual* age begun—duration—interval—regularity—pain—amount—date of 1st period *Operations* see below *Marital* health and compatibility of husband—number of children—health or cause of death of each—abortions—miscarriages—labors (instruments tears time in bed)—fever *Menopause* date and duration—nervous symptoms—discharge since? *Vaginal discharge* color (bloody yellow green white mucus)—time—duration—irritating—warts—tumors

**Male Genitourinary** Gonorrhea—chancroid—chancre—secondary and tertiary symptoms—bubo—potency—frequency—masturbation—promiscuity—health of wife and children

**Allergic** Hay fever—asthma—rashes—migraine—polyuria—cramps—diarrhea—relation to foods furs pollens drugs season—skin tests—anaphylactic reactions

**Injuries** *Character*—parts affected—treatment

**Operations** Dates—disease (how long ill before)—surgeon—hospital—time in bed—drainage—infection in wound—hernia through wound—effect on symptoms—new symptoms

once by hand. This is better than a typewritten sheet dictated from memory.

The important findings on physical examination and the indications for and interpretation of the results of instrumental and laboratory examinations are described in the introductions to the chapters covering the parts of the gastrointestinal tract to which they apply. For detailed descriptions of the techniques of the various laboratory examinations the reader should refer to textbooks on clinical pathology and roentgenology.

## SUMMARY

Thus far the discussions have been devoted essentially to a consideration of the information necessary to arrive at a diagnosis. Some conclusions can be summarized:

1 It is helpful to consider the whole gastrointestinal tract as a single functioning organ and it is necessary to understand both its normal and its disturbed physiology.

2 The symptoms of gastrointestinal disorders are due to disturbed physiologic processes.

3 The normal gastrointestinal tract acts normally when it contains food.

4 An empty gastrointestinal tract acts abnormally. An empty stomach undergoes hunger contractions felt as pangs; ordinarily, as pain with ulcer. An empty intestine does not show normal peristalsis and is apt to become distended and later spastic. Infrequent feedings, especially of fat, tend to cause gallbladder stasis, a forerunner of stones.

5 A disease in any part of the gastrointestinal tract causes disturbed physiology and the symptoms may or may not be characteristic.

6 The diagnosis of diseases of the gastrointestinal tract requires a thorough, careful study, including a careful history, complete physical examination and certain indicated instrumental examinations.

7 No patient should be treated unless a diagnosis has been made. Many lives are lost through symptomatic treatment.

## Treatment

### GENERAL DISCUSSION

*Treatment of any patient without having established a diagnosis is not only inadvisable but also often dangerous.* There was a time when conscientious doctors frowned upon empiricism, symptomatic treatment, the use of "shotgun" mixtures. Today we see more empirical treatment than was done for a couple of generations before the wonder drugs came out. Fifty years ago prescriptions for long lists of drugs (mostly containing quinine and potassium iodide), various local applications

**Head and Neck** *Hair and Skin* color—prominent blood vessels—paralyses—deformities—stigmata *Eyes* appearance—vision—reflexes—hemorrhages—inflammations *Ear Nose Throat* infection—discharge—obstruction—deformities *Mouth* tongue—mucosa—teeth (infections cleanliness missing caps bridges plates)—x rays *Glandular enlargements*—thyroid—pulsations

**Chest** General appearance — shape — expansion — deformities *Breasts* *Lungs* general examination—palpation—percussion—auscultation etc *Heart* size—murmurs—compensation *Arterial System* aneurysm—pulse—blood pressure

**Abdomen** Shape—costal angle—flabbiness—muscles—spasticity—protrusions — hernias — scars — veins — rashes — discolorations — glands — reflexes—fluids *Tenderness* location—reflected pain—degree *Rigidity* degree—location—persistence *Tumor* location—relation to other structures—mobility—consistency *Ptoses* organs mapped out—degree—reduction

**Rectum and Sigmoid** *Digital* sphincter—piles—fissures—fistula—stricture—prolapse—prostate—tumor—bleeding *Proctoscopic* mucosa—tumors—strictures—exudate—smear or biopsy

**Back and Extremities** Deformities—tenderness—discolorations—scars—rashes—infection—varicosities—edema—reflexes—tremors—paralyses—anesthesias etc

## 5 INSTRUMENTAL EXAMINATIONS

Esophagoscopy—bougies—gastroscopy—proctoscopy—peritoneoscopy

## 6 LABORATORY TESTS

Fractional test meal—biliary drainage—blood—serology—stools—urines—functional tests

Note that in this outline words only are used to indicate the questions to be asked and the examinations to be made. Longer more detailed outlines take longer to read and are more difficult to follow. Most clinicians at some time or other have tried submitting questionnaires to patients to be brought to the doctor's office all filled out. This sounds like a good scheme but I have found that even the most intelligent patients even professional people have difficulty in understanding the questions and the inadequately filled in questionnaire is more trouble than a personal history. Another method is one in which the doctor uses a printed form which leaves uniform spaces for the patient's answers. When sometimes a series of questions answered No can be written down in a few words space is wasted and when one answer requires a half page or more it encroaches on other questions so that the result is confusing. I prefer a plain sheet of letter paper with all findings written down at

procedure should be carefully explained to the patient so that his co-operation will be obtained. Methods of treatment include prophylactic physiologic dietetic hormone endocrine medicinal surgical and radio therapeutic measures.

### PROPHYLACTIC TREATMENT

Prophylactic treatment is most important but unfortunately is often not adequately carried out even though it is pointed out to the patient that its neglect has caused his trouble. Included would be the following: *General hygienic care* including adequate exercise rest recreation sun shine and sexual activity.

*Attention to sanitation* including water supply sewage disposal food handlers meat and other food inspection insect control and inspection of gas facilities to mention the most important.

*Focal infections* should be avoided or if present eradicated as follows:

1 Adequate *dental care* including extraction of nonvital and pyorrheic teeth impacted or unerupted teeth and retained root fragments or infected areas. Pyorrhea may be prevented or if present fairly well controlled by thorough and repeated scaling. Though many persons in whom such dental treatments have been neglected may go on for years without untoward effects it is safer in general to prevent trouble by having treatments carried out.

2 Careful treatment or if possible eradication of upper *respiratory infections* with removal of tonsils which are at all suspicious.

3 Prevention of *pelvic infections* during pregnancy and parturition and elimination of any chronic infective process in the cervix uterus or adnexa.

4 Adequate treatment for *venereal diseases*.

5 Removal of any *chronic infections* elsewhere especially rectal.

*Dietetic care* which involves the following of a normal well balanced diet of proper caloric value at regular intervals. Avoidance of contaminated foods and of foods to which a person is allergic is also important.

*A normal bowel habit* as regards time of defecation responding to the urge avoidance of laxatives enemas and suppositories. Normal bowel action should be accomplished by adequate bulk in the diet sufficient water to keep the stool soft and exercise of the abdominal muscles. *Attention to general diseases* hereditary or acquired which might affect the gastrointestinal tract.

*Immunization* including vaccines serums or other measures to prevent disease.

Study of early *symptoms* of gastrointestinal disorders to prevent complications or to detect early malignancies.

*Avoidance of transfusion reactions* can also be included under pro-

enemas stupes cathartics and other measures were freely prescribed for almost any ailment. Today we see an even more careless type of therapy. Some doctors, ordinarily in excellent standing, have been known to prescribe—even over the telephone and without attempting a diagnosis—dangerous drugs such as corticosteroids, antihypertensives, sulfonamides, antibiotics, antihistamines, anticonvulsants, anticholinergics, newfangled cathartics and various types of physical therapy and psychotherapy.

If they fail to be relieved of symptoms, patients are referred to surgeons, a few of whom will do exploratory operations without attempting to make preoperative diagnoses. I am regularly consulted about patients who have had bowel perforations after cortisone, perforated peptic ulcers after rauwolfia preparations, jaundice after chlorpromazine, blood dyscrasias after sulfa drugs, severe colitis after antibiotics, diarrheas after antihistamines, ileus after anticholinergics, intestinal disturbances of various types from cathartics and enemas, to mention but a few. And the tragic fact in most of these cases is that no diagnosis was made before treatment and the patient, in addition to some gastrointestinal or cardiac lesion, has had a new disease added by the ill advised medication. I have seen patients with normal hearts invalidated for years because of "anginal pains" due to gallstones. I have seen patients who were treated with physical therapy for "bursitis" or "spinal arthritis" who had peptic ulcer or gallstones, and patients who had been treated by psychiatrists with psychotherapy and even shock therapy when a large cancer could be palpated in the abdomen. And most tragic of all, I see patients who have been subjected to surgery by two "knife happy" operators (not true surgeons) who have completely overlooked the presence of cancer, gallstones, hepatic cirrhosis and other lesions while doing some other operative procedures.

I have taken up all this space to emphasize the need for a complete diagnosis before treatment is begun. A narcotic for pain may please the patient but may cause his death; an antibiotic for fever or an anticholinergic for spasm may mask the symptoms of a perforation. So, at the risk of offending the patient, it is better not to give any drug until a diagnosis is made. Placebos, such as applications of heat or cold to the abdomen, head or feet, or a hypodermic injection of sterile water, may save the life of a patient with an acute condition of the abdomen. In a chronic case a bland diet, a mild sedative or an oil retention enema will satisfy a patient while a study is being carried out and will usually do no harm.

When the diagnosis has been established, it is good practice to explain to the patient exactly what has been found, using pictures to illustrate the lesions. In cases of malignancy, it is usually better to speak of ulcer or tumor or obstruction and not to mention cancer. A near relative should be told in such a case. The proposed treatment and the reasons for each

procedure should be carefully explained to the patient so that his co-operation will be obtained. Methods of treatment include prophylactic physiologic dietetic hormone endocrine medicinal surgical and radiotherapeutic measures.

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phylaxis Transfusions should be avoided if possible since too many reactions occur in spite of the greatest care in typing preservation and injection Besides this no method has yet been devised to prevent transmission of infectious (homologous serum) hepatitis through transfusion It is well to remember that though transfusion is often a life saving procedure it is a serious operation not to be carried out indiscriminately

### PHYSIOLOGIC TREATMENT

Physiologic treatment refers to any measures which aim to maintain or restore normal physiologic processes and to eliminate any factors which may adversely affect these processes Included would be many of the measures mentioned under Prophylactic Treatment Since the principal functions of the gastrointestinal tract are digestion and absorption of ingested food and elimination of waste products it follows that the most important consideration in physiologic treatment must be attention to diet

In order to prescribe a diet which will produce a definite therapeutic effect the clinician should have at hand a simple book on dietetics The many large books on nutrition are too lengthy and go into details which are interesting mostly to other nutritionists With a little practice in calculating caloric values of the usual portions of common foods and in general their content of essential ingredients it soon becomes fairly easy to prescribe a properly balanced diet for an individual patient

### DIETETIC TREATMENT

It is most important in all cases to explain to a patient what a normal diet should consist of wherein his diet has been faulty and why the diet prescribed will help his condition After such a briefing we can hope for a patient's full cooperation in following a diet It is careless and inefficient to prescribe a diet in such general terms as take a bland diet or void roughage or cut down on fats The patient is confused and dietetic cure is not accomplished Another mistake is to give the patient a printed or typewritten diet obviously not designed especially for him He feels that he could have obtained such a prescription by reading a health column The most pernicious form is one which lists only foods to be avoided without any diet prescription Even if a list of foods allowed is added the patient still does not know what to eat In order to avoid any misunderstanding it is best to write down in detail exactly what the patient is to eat at each meal and between meals how much water is to be taken and what additional treatment is being prescribed

For a diet card I use a plain white card size 4 1/2 by 5 1/2 with my name address and telephone number at the top to make it easy for the patient to call me about his diet As a heading I use the words "Diet for

"leaving a blank space for the patient's name. Beneath this is a note "Eat everything on this list—nothing not on it." Figure 3 shows such a card filled out for a normal diet. This will be used as a pattern for other diets with variations in the ingredients according to need.

In prescribing a diet it is important to realize that food is used in four ways in the treatment of a patient: (1) Food is necessary, of course, for adequate nutrition. (2) It is important for proper functioning of the gastrointestinal tract. (3) It can be used for its mechanical effects on the gastrointestinal tract. (4) Its caloric value must be adapted to individual requirements.

Diet for _____		
Eat everything on this list—nothing not on it		
Eat whole grain breads and cereals		
<i>Breakfast 8</i>	<i>Lunch 1</i>	<i>Dinner 6</i>
Cereal—cooked or dry (1 cup with milk and sugar)	Chicken, egg or meat Vegetables—cooked (1 cup)	Meat or fish—3-4 oz (or egg or cheese)
Eggs—1 or 2—soft	Salad	Vegetables—cooked (1 cup)
Bread and butter (or toast)	Bread and butter	Salad
Milk (1 glass)	Milk (1 glass)	Bread and butter
Fruit—raw	Desert—fruit	Desert—fruit
<i>Between meals and at bedtime</i> Gelatin (1 envelope mixed in 1 glass of water) and 1 glass of milk or fruit juice with or without crackers, bread or cake		
<i>Water</i> At least 4 to 6 glasses a day		
<i>Vitamins and minerals</i> As directed on back		
<i>Avoid</i> Irritants, alcohol, coffee, tea, condiments and laxatives		

Figure 3 Diet card filled out for normal diet

## Adequate Nutrition

Adequate nutrition requires what is commonly called a balanced diet. For nearly a century nutrition experts have been conducting nutrition and diet surveys in order to determine what constitutes a balanced diet, what proportions of proteins, fats, carbohydrates and other vitamins and minerals and caloric values would be ideal for normal nutrition. Too long they tacitly assumed that the human race consists of individuals who have average needs for these food elements. This has been shown to be far from true.

Each individual organism that has a distinctive genetic background has distinctive nutritional needs to maintain normal health. Environmental differences, seasonal influences, the amount of exercise taken, and various anatomical, compositional, enzymic, endocrine, and excretory factors are some of the conditions which account for variations in diet require-

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Each individual organism that has a distinctive genetic background has distinctive nutritional needs to maintain normal health. Environmental differences, seasonal influences, the amount of exercise taken, and various anatomical, compositional, enzymic, endocrine, and excretory factors are some of the conditions which account for variations in diet require-

ments In addition to this disease causes marked changes in nutritional needs

Apart from the varying requirements of the human organism there is the problem of varying composition of foods The proportions not only of vitamins and minerals but also of proteins fats and carbohydrates vary according to the health of the animal or plant from which they are obtained according to their diets (foods fertilizers soil water) according to weather conditions during their lifetime and many other factors It is therefore practically impossible to determine exactly either the nutritional needs of a given individual or the constituents of any diet

In order to prescribe an adequate diet instead of adhering to a set of rules regarding the proper proportions of each of the food elements or to allowances based on grams per kilogram of body weight it is more simple to calculate approximately the quantity of each that is considered optimal and to prescribe accordingly With the normal diet outline above as a standard there follows a brief discussion of each essential ingredient and the ways it can be modified in disease

**Protein** Protein is the most important food factor It is needed to maintain growth and repair of tissues to replenish protein stores and to maintain normal endocrine function especially that of the gonads As proteins contain various combinations of amino acids it is desirable to consume various foods containing protein in order to get all required amino acids The amino acid glycine most common in gelatin improves physical performance Gelatin is also of value to produce very fine curds in milk when it encounters gastric juice Food surveys have resulted in recommendations that from 60 to 100 gm of protein a day will provide all proteins necessary for a healthy adult the amount to be modified by sex age exercise and body build and weight

The normal diet outlined above (p 21) with average portions of each ingredient and three envelopes of pure powdered gelatin would average about 125 gm of protein a fairly high amount By prescribing more or less of the protein rich foods such as eggs meat gelatin milk and cheese the total protein content can be easily modified In the diet mentioned taking out the between meal feedings reduces the protein to 75 gm Protein is usually given by ingestion although solutions of amino acids casein hydrolysates and normal serum albumin can be used intravenously when required providing up to 50 to 75 gm of protein a day

**Fat** Fat was formerly considered of no importance and often was ruthlessly cut out of diets for reducing weight for avoiding arteriosclerosis and for the treatment of biliary tract diseases In addition to its use as a fuel fat is essential for adequate liver function for absorption and elaboration of fat soluble vitamins for adequate functioning of the pituitary gonads thyroid and adrenals and for the stimulation of biliary tract function No definite evidence has yet been adduced to

prove the vague theories that ingested fat especially animal fat adds to the likelihood of the development of arteriosclerosis or of gallstones. Cholesterol deposition can be greatly reduced if oils containing the unsaturated essential fatty acids are added to a diet even though considerable amounts of animal fats containing the saturated heavier fats are present in the diet. The essential fatty acids play an important part in growth in skin metabolism in liver function and in maintaining a healthy vascular system. It is wise to include the essential fatty acids in any diet. The oils richest in these acids are corn, peanut and soybean oils and to some extent cotton seed oil. An adequate diet such as the one illustrated above should contain about 100 gm of fat. By modifying the quantities of fat rich foods such as milk, eggs, cheese, butter and the vegetable oils, the amount of fat prescribed can be changed. Fat must be given orally although recent experiments with intravenously administered emulsions have apparently shown good results, such injections are dangerous in liver diseases however.

**Carbohydrates** Carbohydrates are of value as fuel, their combustion not as rapid as that of fat, being more prolonged. Carbohydrates also favor neuromuscular coordination, the capacity for muscular work and acuity of vision in dim light. The carbohydrates not used as fuel and too great in quantity for all of it to be stored in the liver and muscles as glycogen are converted into fat. Excessive ingestion of carbohydrate is the most common cause of obesity. The quantity of carbohydrates needed per day cannot be stated categorically but enough must be consumed to provide adequate calories.

The sample normal diet outlined above contains about 175 to 200 gm of carbohydrates. Through the use of foods of varying carbohydrate content diets can be modified as required. Carbohydrates are usually given by mouth and in the form of whole grain cereals and fresh fruits and vegetables provide in addition more or less adequate supplies of vitamins and minerals. The pure carbohydrates dextrose and levulose can be given parenterally for rapid effect and can be safely used in 5 or 10 per cent solutions in water or in normal saline solution as required.

**Vitamins** Vitamins are organic compounds, some fat soluble others water soluble which are required for normal growth and maintenance of the life of animals including man. Man is not able to synthesize these compounds except two, vitamin D which is formed in the body by the action of sunshine on the skin and vitamin K which is synthesized in the intestines. Practically all the vitamins are obtained from animals and plants which synthesize them. Vitamins act as catalysts, only minute amounts being necessary to produce important effects. Some are closely related to hormones both in composition and performance and may stimulate or supplement their action. Many vitamins are now being pro-

duced synthetically and are identical with those obtained from natural sources. The liver is an important reservoir for vitamins.

Innumerable experiments in animals and man have been carried on over the years and new effects and uses of vitamins are regularly discovered. It is unfortunate that frequently a fragmentary knowledge of vitamins has resulted in grave errors in therapy. When a vitamin is discovered which has an effect on some bodily function there has been a tendency on the part of doctors to prescribe that single vitamin for any disturbance of such function and in very large doses. Many patients have been harmed instead of helped by such overdosage.

*Vitamin deficiencies* may be due to insufficient quantities of vitamins in the diet or to their faulty absorption because of disease as in ulcerative colitis sprue and other diarrheal diseases. *Deficiency disease* may be caused by direct disintegration of tissues because of the absence of their hormone like action or to secondary infection as a result of lowered resistance. *Infections* of any kind deplete vitamin storage so that larger doses of vitamins are required.

*Allergic reactions to vitamins* have been described. Whether the reactions are due to sensitivity to the vitamins themselves or to the animal or plant from which they are obtained has not been adequately determined.

In *prescribing vitamins* it is important to be guided by certain facts which have been discovered as a result of extensive nutrition studies over the past three or four decades as follows:

1. It is difficult if not impossible to prescribe a diet which in itself will contain an adequate supply of vitamins. The variations in the content of vitamins even in different batches of vegetables and fruits from the same field or orchard and the variations in animals depending upon food and environment have already been mentioned.

2. Vitamin supplements are a valuable addition to any diet even though no gross evidence of vitamin deficiency is present.

3. Even though a patient shows definite evidences of one specific vitamin deficiency it is known that rarely if ever is there deficiency of only one vitamin. It is wise to prescribe a mixture of vitamins instead of only the one indicated.

4. Vitamins supplement each other some losing their efficacy in the absence of others.

5. Overdosage of any vitamins is to be avoided as much as possible although in rare cases very large doses may produce a pharmacologic rather than a vitamin effect.

A brief résumé of essential information about each of the most important vitamins follows.

*Vitamin A* a fat soluble vitamin is necessary for maintenance of normal mucosal epithelium and tooth structures and for visual acuity. Deficiency results in epithelial atrophy with susceptibility to infection of the

mucosa of eyes nasopharynx and gastrointestinal and genitourinary tracts. Eye findings include night blindness and xerophthalmia. Vitamin A is mainly stored in the liver which regulates its concentration. It is most plentiful in fish liver oils, eggs, most animal fats and in vegetables (green leafy or yellow) containing carotene, the precursor of this vitamin. The vegetables containing most are peas, spinach, carrots, kale and lettuce, but the human body utilizes their carotene in varying amounts. The average normal requirement is estimated at 5000 to 10,000 U.S.P. units per day. In old age the requirement is considerably higher. Our normal diet contains over 20,000 units. Any diet containing plenty of milk and pigmented vegetables will contain ample vitamin A. In diets containing insufficient quantities of these foods the vitamin can be added in the form of fish liver oil or vitamin concentrates. When parenteral feedings are being given, this vitamin is now available for such use in aqueous solution.

**Vitamin B complex**—a mixture of at least a dozen different vitamins, all water soluble, each with its own function in the body, occurs in its natural state in high concentration in yeast and liver. Some of the individual vitamins also occur in other foods, particularly in whole grains, meat (especially pork), eggs, nuts, legumes and most vegetables. The most important B vitamins are the following:

**Vitamin B<sub>1</sub>** (thiamine or thiamine hydrochloride) has been called the antiberiberi vitamin. Its beneficial effect in neuritis from any cause, even in alcoholic neuritis, and in pellagra and in so-called beriberi heart is marked. It will often overcome anorexia of dietary origin. As a co-factor with magnesium it improves carbohydrate metabolism and promotes growth. It occurs in all the foods mentioned above and has been produced synthetically. The normal requirement for an adult is supposed to be 2 to 2.5 mg. a day, but it is safer to provide from 3 to 5 mg. In our normal diet there are at least 4 or 5 mg. of thiamine. In cases of need the quantity of thiamine can be increased by the addition of wheat germ oil, rich also in other B complex vitamins. The purified vitamin in doses of 50 to 100 mg. has been given without harm.

**Vitamin B<sub>2</sub>** (riboflavin) promotes growth, appetite and general health. Deficiency causes cheilosis, keratitis, glossitis and photophobia. It occurs in all the substances containing thiamine, but is also present in milk and cheese. The standard requirement is placed at 2 to 4 mg. a day. Our normal diet contains about 2 mg. When symptoms of deficiency are present, riboflavin is available in vitamin tablets. Up to 15 mg. a day can do no harm.

**Niacin** (nicotinic acid) promotes tissue respiration, gastrointestinal function and normal skin, and is essential for growth and general health. Deficiency produces pellagra, with its skin, gastrointestinal and nervous manifestations. Nicotinic acid occurs in all the foods mentioned under B.



complex The average requirement is from 15 to 25 mg a day Our normal diet contains between 15 and 20 mg Nicotin is available in tablet form In cases of deficiency up to 500 mg has done no harm

*Vitamin B<sub>6</sub>* is really a group of closely related chemical compounds related to *pyridoxine* It is an essential nutrient playing an important part in amino acid metabolism Deficiency is unusual but appears to cause muscular weakness Parkinsonism muscular atrophy and dystrophy have been attributed to this cause as have epileptiform seizures in children Seborrhea like skin lesions neuritic manifestations and vomiting of pregnancy have also been attributed to such deficiency This vitamin has been produced synthetically but occurs normally in adequate amounts in many common foodstuffs as it does in our normal diet The average requirement is supposed to be 1.5 to 2 mg Up to 100 mg have been given without bad effects

*Vitamin B<sub>12</sub>* (cyanocobalamin) is essential for normal growth and nutrition It is concerned with hematopoiesis and with maturation of epithelial cells (gastric and oral) It has definite neurotropic and lipotropic effects It contains cobalt Deficiency results in pernicious anemia and its manifestations Its source in nature comes from metabolic activity of microorganisms especially of the alimentary tract of animals Bound to protein it is inactive until split from it by digestion It is found mostly in liver kidney and heart of beef and lamb and in clams Only small amounts are found in beef muscle pork fowl and egg yolk Its dosage in pernicious anemia achylia gastrectomy hepatic insufficiency intestinal anastomoses and malabsorption syndrome is 1 or 2 micrograms a day given intramuscularly It is not as effective given orally

*Vitamin B* (folic acid) is another antianemia factor also part of the B complex It occurs in the same foods as other B vitamins and is also synthesized commercially It is used for preventing and treating pernicious anemia in doses from 0.5 to 10 mg a day and is usually given together with vitamin B<sub>12</sub>

*Pantothenic acid* (calcium pantothenate) appears to be essential for normal gastrointestinal function carbohydrate function and adrenal function It also promotes normal skin and hair It occurs with other members of the B complex No definite optimal amount has been discovered although it is usually prescribed in doses of 2 to 10 mg a day

*Choline* and *inositol* are necessary for the formation and transport of phospholipids and nourish bone marrow and conjunctiva Deficiency results in fatty infiltration of the liver hypercholesteremia and hyperlipemia Such deficiency is due to food deficiency in general but especially B complex deficiency These vitamins are found in milk cheese and vegetables In severe hepatic insufficiency 2 to 4 gm of choline and 0.5 to 1 gm of inositol are helpful

Other members of the B complex vitamins are being studied and uses

will undoubtedly be found for them. This is used as an argument for administration of the whole natural complex in the form of wheat germ.

**Vitamin C** (ascorbic acid) the antiscorbutic vitamin, water soluble, is essential for maintenance of collagen, the intercellular substance of teeth, bones, skin and blood vessels. Deprivation results in frank scurvy or in some of its manifestations, such as hemorrhages, dental caries, gum recession and bleeding, anorexia, anemia, poor wound healing and tendency to infection. It occurs in fruits and vegetables, particularly in citrus fruits, raw cabbage, tomatoes and leafy vegetables in general. As it is destroyed by heat, it can be counted upon only in raw vegetables and fruits. The synthetic has the same effect as the natural vitamin. The body requires at least 100 mg. a day. In deficiencies it can be increased by more raw foods or by the addition of synthetic ascorbic acid, which may be given by mouth or parenterally. Doses up to 1000 mg. a day are easily tolerated. It is useful in the treatment of collagen diseases.

**Vitamin D** (calciferol, activated cholesterol), oil soluble, the antirachitic vitamin, is essential for the deposition of calcium and phosphorus in teeth and bones. Deficiency produces rickets and tetany in children, osteomalacia in adults. It is abundant in fish liver oils, also present in eggs, milk and butter, and is formed in the body when the skin is exposed to sunlight. It is commercially produced by irradiation of ergosterol and is added to milk by most dairies. It is also marketed in an aqueous solution. The requirement is about 1000 to 1500 units a day, somewhat higher past middle life. In excessive doses, 5000 or more units a day, it may cause hypercalcemia. In cases of deficiency, cod liver oil and other foods can be increased or the vitamin can be given in capsules.

**Vitamin E** (alpha tocopherol), fat and water soluble, the antisterility vitamin, appears to be concerned in the maturation and differentiation of certain cells, especially on spermatogenesis, the fertilized ovum, muscle and nerve tissue and probably on some glands of internal secretion. Deficiency is said to result in habitual abortion, sterility, muscular dystrophy, fibrositis and some forms of peripheral vascular disease. It occurs in various oils, such as those obtained from wheat and rice germ, corn and cotton seed, and in lettuce and alfalfa. The optimal body requirement has not been proved, but the vitamin has been used with favorable results in the conditions mentioned above, in doses of 40 to 150 mg. a day. Overdosage apparently does no harm.

**Vitamin K** (menadione), the antihemorrhagic vitamin, fat soluble, is essential for prothrombin formation and therefore for normal blood coagulation. Deficiency produces prolonged prothrombin and bleeding time and tends to produce hemorrhages. It is normally synthesized in the intestine and also artificially. A good diet usually contains a sufficient amount. In patients with hemorrhages from any cause, but especially when prothrombin time is prolonged, as in jaundice or after the adminis-

tration of heparin or Dicumarol menadione can be given parenterally or by mouth in doses of 2 to 5 mg a day. For prophylaxis 1 mg a day is used.

These are the only vitamins definitely shown to be required by the human body. Suggestions are constantly being made regarding the possibility of vitamins being responsible when an investigator finds that some specific food apparently has been beneficial in the treatment of some disease. Ulcer vitamins, lactation vitamin, a capillary vitamin, a bacterial vitamin and others have been proposed but are not yet proved to be of specific value.

*Bioflavonoids* sometimes called vitamin P are usually not classed as vitamins but play an important role in the utilization of vitamins especially vitamin C. They are most abundant in orange pulp and a deficiency occurs when orange juice is strained. They can be given in capsule form about 100 mg a day being the usual dose.

*Minerals* Minerals are just as important as vitamins for the maintenance of normal health and well being. Some vitamins are effective only in the presence of minerals as in the case of vitamin D and calcium and vitamin B<sub>1</sub> with phosphorus. Minerals present in food get there as a result of absorption from the soil by plants and the consumption of these plants for food by animals. Some of the essential minerals are required in fairly large amounts such as calcium and phosphorus others only in very small quantity these being called the essential trace elements. The mineral content of soil varies greatly not only in various parts of the world but even in different areas on a farm. When repeated crops have been grown in a given soil with no replacement of minerals great depletion may result so that the plants and animals which eat them will be deficient in some minerals. It is therefore safer as a prophylactic measure to add some minerals to a normal diet even though it contains foods which are supposed to contain all essential minerals.

**PROPORTIONS OF MINERALS** *The proportions of the minerals is important* for excess of one may antagonize another and actually cause disease. Thus an imbalance of calcium and phosphorus may produce rickets whereas a proportion of 1 to 1.5 made up of 0.6 gm of calcium and 0.9 gm of phosphorus a day will cure the disease but then only when accompanied by sufficient vitamins A and D. Ordinarily adequate calcium and phosphorus are provided in a balanced diet containing sufficient milk and cheese. They can be added as supplements.

*Sodium and potassium* are similarly related. An excess of sodium as for instance in Cushing's syndrome causes a deficiency in potassium. Potassium deficiency must be suspected in any condition involving disturbance of electrolytes or body water. The use of excessive amounts of normal saline solution parenterally is dangerous from this standpoint.

and some potassium is always required under those circumstances. Ordinarily a normal diet will provide these elements in sufficient amount.

**TRACE ELEMENTS** Of the essential trace elements iron, copper and cobalt are essential for the formation of hemoglobin. The copper does not enter into the formation but acts as a catalyst. The body need for iron is from 5 to 15 mg. a day; of copper, one tenth as much. The cobalt of which less than 0.1 mg. a day is ample, is a constituent of vitamin B<sub>12</sub> which is essential for regeneration of red blood cells. These minerals are present in a balanced diet but are used as adjuncts in anemias in minute doses. Manganese has also been supposed to have an effect in anemias but this is questionable. Its dosage is 1 mg. a day. Molybdenum has recently been added to the list of hematinics. Its dosage is 2 to 3 mg. a day.

Zinc, useful in anemias in amounts of 0.2 to 0.5 mg. a day, will cause anemia if taken in excess. Magnesium is important in carbohydrate metabolism acting with thiamine. It activates phosphatase. A deficit is found in severe diabetes. It may produce tetany. One milligram a day is required. Iodine is essential in the synthesis of thyroxine in the body. A deficiency of iodine results in goiter. Iodine is present in adequate amounts in salt water fish and in plants raised on or near the sea coast. Inland it is necessary to take iodine as a supplement usually as iodized salt. Consumption of large amounts may be definitely harmful. Fluorine has recently been advocated as a preventive of tooth decay. It exists in the water supply of some regions and is sometimes added to drinking water in those deficient. One part in one million parts of drinking water is the optimal dosage. More than one and one half parts may cause darkening of the teeth and bone changes.

**SUMMARY** It will be seen from the discussion of the roles of vitamins and minerals in nutrition that the use of multivitamin and multimineral preparations is not "shot gun" treatment but is on a strictly scientific basis.

### Effects of Diet on Gastrointestinal Function

Normal function of any organ is best maintained by exercise of that function. This is true particularly of the gastrointestinal tract. Starvation and even failure to eat a sufficient quantity and variety of foods produce changes in function for which an adequate diet is the best remedy. Secretion, absorption and motility are all affected. As a result of having little or nothing to digest the digestive glandular structures will become diminished in number and effectiveness, the mucosa will begin to atrophy, interfering with absorption, and motility will be disturbed with resultant atony and a tendency to ileus or intermittent spasms. Constipation is a natural result. Neglect of fat in the diet by failing to produce the relaxation of the sphincter of Oddi and the contraction of the gallbladder will produce biliary stasis and favor the formation of gallstones. Pancreatic

secretion and hepatic secretion are also disturbed. It is therefore important in planning any diet for whatever condition it is prescribed to be sure that each meal contains the foods necessary to stimulate all these functions. This is best obtained by the balanced diet previously mentioned.

If in disease some functions are overstimulated or understimulated the diet can be modified in such a way as to influence these effects. In gastric hypersecretion stimulation to secretion can be reduced by the avoidance of meat and meat extractives, caffeine containing drinks, condiments and spices and frequent small feedings of less stimulating foods as in an ulcer diet. In gastric achylia in which the protein envelopes of meat fibers and starch granules are not digested meats should be avoided for a while and later thoroughly cooked as starches should be in order to burst the envelope. In gallbladder disease frequent feedings of fats will promote biliary drainage. In pancreatic achylia easily digestible foods with pancreatin added are indicated. In colonic hypermotility a low residue diet may be used to reduce stimulation to motility or a high residue diet to provide bulk for normal colonic motor function.

### The Mechanical Effects of Foods

In the gastrointestinal tract the mechanical effects of foods must be considered in prescribing a diet. These effects would include soothing or irritating reactions and response to thin liquid or thick and firm bulky or nonresidue hot or cold substances. A *soothing or bland diet* used to soothe an irritated or ulcerated mucosa would consist largely of milk, eggs, cereals, puddings, gelatin and ice cream taken frequently to maintain the soothing effect. An *irritating diet* not usually advisable might be used to stimulate sluggish musculature or glands and would contain spices, condiments or coffee or other caffeine containing drinks. *Thin liquid diets* would be required when there is a definite narrowing of the lumen of the esophagus, stomach, duodenum or even further down. Such diets would include milk, raw eggs, gelatin, cream and dextrose taken frequently and in quantities sufficient to provide adequate caloric value. *Thick firm foods* useful in causing more forceful peristalsis to push material through a spastic area as in cardiospasm would consist of bread, stuffs, vegetables, fruits and meats. *Bulk producing diets* useful to provide sufficient colonic content to stimulate bowel action would consist of extra amounts of indigestible residue, celluloses, leafy vegetables, skins and seeds and pulp of fruit and hulls of grains. *Nonresidue diets* used to prevent as much as possible any extra amount of material in a colon which is irritated, inflamed or narrowed would consist of a liquid diet with added fully digestible cereals, vegetables, fruits and possibly some meats. Excessively hot as well as cold foods should be avoided, excessive

heat causing marked esophageal and gastric irritation cold often stimulating peristalsis to the point of causing diarrhea

### The Caloric Requirements of a Diet

These requirements are subject to wide variations being governed by sex age size body habitus amount of exercise occupation heredity, disease and other factors A study of recommended caloric values for diets for the average person shows wide variations in the tables published by different authorities and but little if any allowance is made for the various factors mentioned Some tables are based on the individual's weight but weight tables also show wide variations when using height body habitus age and sex as a basis Besides this the tables take into consideration only the caloric value of the constituents of the diet and fail to indicate that this differs materially from the number of calories which after being processed by the gastrointestinal tract are absorbed and utilized by the body It is obvious then that tables of caloric requirements are of only limited value In general the only practical guide to the caloric value of a diet and the caloric requirement of an individual is careful observation of weight fluctuations and general health

**High Caloric Diet** High caloric diets are indicated as follows (1) when weight is below normal from any cause (2) in conditions accompanied by increased metabolism such as a febrile disease hyperthyroidism strenuous exercise (3) preceding operations or any period of expected fasting or low food intake

**High caloric foods** including carbohydrates in the form of sugars syrups cereals breadstuffs and high carbohydrate vegetables and fruits can be added to a normal diet with and between meals or they can form a part of any type of otherwise restricted diets even liquid diets Fats and oils also furnish extra calories In liquid diets the fat is simply furnished by cream the carbohydrate by dextrose Protein and the essential vitamins and minerals must not be neglected in either high or low caloric diets

**Low Caloric Diet** Low caloric diets are indicated (1) when weight is above normal (obesity) (2) in conditions accompanied by a lowered metabolism such as hypothyroidism or an inactive life

In a low caloric diet sweets and fatty foods must be reduced to a minimum low caloric vegetables and fruits must be prescribed and high caloric foods especially rich desserts gravies syrups and alcohol must be interdicted It must be remembered however that some fat at least 50 to 75 gm a day is required to maintain biliary tract function and prevent gallstones Salt should not be avoided but should be used in moderation

For obesity a diet like that shown on page 32 will be satisfactory if strictly adhered to in the average obese patient Medication such as

thyroid or amphetamine preparations may cause marked endocrine imbalance and should be avoided

## Reducing Diet

### Breakfast

2 eggs boiled or poached  
Bread one thin slice preferably protein bread  
Butter one medium pat  
Milk whole 4 ounces (1 glass)  
Fruit raw

### Lunch

Meat or fish (4 ounces) or cheese (cottage  $\frac{3}{4}$  cup American cheese 1 inch cube)  
Vegetables from list cooked (1 cup)  
Salad with dressing of corn soybean or peanut oil and vinegar or lemon juice  
Bread and butter as at breakfast  
Milk (4 ounces) 1 glass  
Fruit raw or canned from list†

### Dinner

Same as lunch

### Between meals and at bedtime

Gelatin (one envelope) in water and milk (1 glass)  
Water at least 4 or 5 glasses a day  
Salt just enough to make food palatable  
Vitamin and mineral capsules daily

*Vegetables and fruits must be restricted to those on the following list of 3 and 5 per cent carbohydrate content*

### Vegetables (plain raw or cooked)

Asparagus	Chard	Mushrooms
Artichokes	Cress	Parsley
Beans—green and wax	Cucumbers	Peppers
Beet greens	Endive	Radishes
Broccoli	Escarole	Rhubarb
Cabbage	Kale	Sauerkraut
Cauliflower	Kohlrabi	Spinach
Celery	Lettuce	Squash
		Tomatoes

### † Fruits (without sugar)

Blackberries	Muskmelon
Cantaloupe	Orange
Cherries	Pears
Grapefruit	Peaches
Honeydew	Plums
Lemons	Strawberries
Limes	Watermelon
Loganberries	

This diet which will average 1500 to 2000 calories will not occasion a rapid reduction in weight but should reduce weight by about a pound or two a week. It is adequate for normal nutrition and may be kept up for long periods without producing undue hunger. If it can be proved that a patient is adhering to the diet and not reducing a moderate reduction in

the quantities of each constituent may be required. When a normal weight is attained the effect of additions to the diet must be carefully checked by weight recording.

Freak "reducing diets" imbalanced and markedly deficient in essential food elements are brought out at regular intervals. Persons following such diets without careful medical supervision are often badly damaged by them. The recent much advertised low protein diet consisting of dextrose, corn oil and evaporated milk providing 900 calories a day is an example of such harmful diets.

### Guides for Formulating a Diet

**Oral Feedings** It is easy to write out a diet on the spur of the moment if the "normal diet" previously outlined is used as a guide. The breakfast can remain practically the same, choosing smooth cereal, fine white bread and orange juice for the soothing smooth diet instead of coarse whole grain cereal, cracked wheat bread and fruit with skin which would be used in a bulk producing irritating diet. For the other two meals when a smooth soothing diet is indicated, eggs or cream, cheese or peanut butter can be used for the protein, the vegetables can be pureed and strained, and the dessert can be a smooth pudding, gelatin dessert or ice cream. For a coarse bulk producing diet the vegetables should be largely of the leafy type with plenty of lettuce, raw cabbage or other raw vegetables and raw fruits for dessert. Methods of regulating the caloric values have been described.

Between meal and bedtime feedings, always desirable, should consist of more milk, cream and dextrose with powdered gelatin (one envelope of the commercial product) in the smooth high caloric diets. The gelatin is difficult to dissolve and is best taken simply mixed with water and swallowed in fifteen or twenty seconds before it becomes gummy.

By using a prescription blank or better a card such as has been illustrated, the diet can be written down while it is being described, and the hours for feedings can be adapted to the patient's occupation or habits. The diets suggested for each of the diseases described in this book will follow the foregoing outlines.

When feedings by mouth are difficult or impossible, it is desirable to provide adequate nutrition by tube or rectal feeding or parenteral alimentation.

**Tube Feedings** Feeding through a small caliber tube such as the smooth tipped small Levin tube, the newer polyvinyl nasogastric tube or gastric or duodenal tubes with metal tips is useful for maintaining nutrition in various conditions. They may be passed through the nose or mouth, preferably the mouth which is less uncomfortable for the patient, especially if there is nasal irritation or narrowing. Such liquid feedings can be given patients (1) who are unwilling to swallow, (2) who have



burns narrowing or spasm in the esophagus (3) who have conditions in which it may be desirable to feed directly into the duodenum (4) who have had operations on the stomach or duodenum when the tube can be passed into the stomach before operation and placed well down in the jejunum by the surgeon. In most cases patients will permit the tubes to remain *in situ* while the feedings are required.

With proper choice of ingredients adequate nutrition can be instituted or maintained. The liquid feedings at a temperature of 105 to 110° F. can be slowly poured into the tube through a funnel 6 to 12 ounces at a feeding every two or three hours or can be allowed to drip into the tube by means of a parenteral drip setup. The great nitrogen vitamin and mineral losses under severe stress especially after operations on the gastrointestinal tract must be considered in planning the nutrients to be introduced.

For *intragastric feedings* a suitable simple formula easily made and easily modified to supply different needs is as follows:

Dextrose	60 gm
Cream (20%)	60 gm
Eggs (2) raw	beaten into the formula
Milk to 1000 cc	

This formula provides about 45 gm of protein 50 gm of fat 140 gm of carbohydrate with over 1100 calories. Eight ounces every three hours will supply over 2000 calories 90 gm of protein 100 gm of fat every two hours over 3000 calories nearly 150 gm of protein and fat.

Vitamins and minerals in the form of one of the commercial preparations in adequate amounts should be added to the formula. There are also on the market various preparations in powder form which will supply adequate nutrients when dissolved in water. Sustagen is an example of such a preparation.

For *duodenal feedings* predigested protein in the form of one of the various amino acid mixtures can be substituted for the egg.

*Rectal feedings* formerly very popular have been all but abandoned. In the old days when regular course dinners were injected into the rectum and later washed out with saline solution to make room for another feeding little if any benefit resulted. Then it was found that water or normal saline solution was absorbed fairly rapidly either by injection of 6 or 8 ounces every six hours or by the continuous drip method. With the advent of intravenous feedings rectal alimentation was practically eliminated.

In the knowledge however that solutions of electrolytes dextrose and alcohol are readily absorbed by the rectal and sigmoid mucosa it is worthwhile to use this method of adding these factors to the circulation in case of dehydration from any cause especially postoperatively. The continuous drip requires such careful observation that it is impracticable

but by the slow instillation every four to eight hours of 6 or 8 ounces of warm solution with the patient lying on the left side knees drawn up and kept quiet for a time as much as a liter of fluid can be given in twenty four hours. A preliminary saline enema may be desirable to empty the rectum but is not necessary when the bowel is known to be empty. A suitable nearly isotonic solution for rectal instillation would be as follows

Dextrose	50 gm
Sodium chloride	3 gm
Potassium chloride	2 gm
Calcium chloride	1 gm
Alcohol	60 cc
Water	q.s. ad L.I

This solution containing over 600 calories is easily prepared and can be modified according to its absorbability as demonstrated by the amount of residue if any remaining in the rectum upon slow careful introduction of the small catheter for the next feeding. It can easily be given at home by an untrained attendant to whom it has been demonstrated. After a few days absorption may be much reduced and the feedings will have to be stopped. Meanwhile the amount of parenteral feedings required will have been reduced or such feedings may even have been obviated. Without discomfort to the patient a period of crisis can thus be tided over. Vitamins and minerals should be given parenterally during rectal alimentation.

**Parenteral Feedings** Intravenous injections of electrolytes and nutrients have largely superseded hypodermoclysis for parenteral feedings although with the addition of hyaluronidase many nutrients will absorb when given by the older method.

**NUTRIENTS** Nutrients in stable and safe solutions for intravenous use are now prepared by many manufacturers. Dextrose levulose amino acids protein hydrolysates and fat emulsions are now available to provide a patient with adequate nutrition. Vitamins and minerals can be added to the solution or may be given intramuscularly to complete the dietary requirements. *With any suspicion of liver damage however the fat emulsions must be avoided since they may precipitate coma and cause death in such cases.*

**WATER AND ELECTROLYTES** Aside from providing adequate food it is generally accepted that regulation of water and electrolyte balance is perhaps the most important factor of parenteral therapy. The proportions of intracellular and extracellular water and electrolytes are constantly changing although kept at a fairly constant balance in health. Their intake in food and drink and their excretion by kidneys skin and gastrointestinal tract minimize any temporary imbalance. The maintenance of water and electrolyte balance can be guided fairly accu-

rately by measuring the entire intake and output of liquids and by various blood studies including cell counts hematocrit levels of proteins albumin sodium potassium chloride carbon dioxide and others. Such studies frequently repeated require hospital care and really expert technicians. Even so the findings are never absolutely accurate. A knowledge of the various causes of electrolyte deficiency and the symptoms due to deprivation of specific electrolytes will form a practical guide to therapy.

The average normal amounts of the principal electrolytes in normal blood are as follows in milligrams per 100 ml.

Sodium	310-345
Potassium	16-22
Calcium	9-11
Magnesium	1-3.5
Chloride	350-500
Phosphorus	3-6
(higher in children)	

*Sodium deficiency* (hyponatremia) may be caused by deficient salt intake, loss of sodium from excessive sweating, urination or gastrointestinal secretions, or an adrenocortical insufficiency. It results in dehydration and reduced urinary chloride excretion which may cause dizziness, nausea, hypotension and later stupor or convulsions. Acidosis is a frequent accompaniment.

*Sodium excess* (hypernatremia) may result from excessive sodium intake by mouth or parenterally from administration of corticosteroids or from congestive heart failure. It may produce tissue edema, headaches and hypertension.

*Potassium deficiency* (hypokalemia) is a result of excessive tissue breakdown, excessive intake of sodium and inadequate intake of potassium. Its most frequent causes are starvation, diarrhea, gastric or intestinal suction or vomiting, shock, trauma (including operations) and loss of fluid from wounds, burns and fistulas. Diseases accompanied by tissue damage such as cancer, acute infectious diseases and nephritis, will also cause losses of potassium and water. Symptoms include a generalized muscular weakness, both of voluntary and involuntary muscles, resulting in weakness of the extremities, muscular twitchings or flaccid paralysis, difficulty in breathing and abdominal distention. Irregular pulse and mental hebetude may occur. Electrocardiographic changes are frequent. Alkalosis is often an accompaniment.

*Potassium excess* (hyperkalemia) may be caused by excessive tissue breakdown accompanied by inadequate urine volume to provide for normal potassium excretion, or it may result from excessive potassium intake. An early symptom is numbness followed by paresthesias of the extremities and general weakness. Electrocardiograms will reveal characteristic changes and heart block may occur.

*Calcium deficiency* (hypocalcemia) is caused by excessive loss of cal-

**crum** It occurs in acute pancreatitis severe infections in subcutaneous tissue or peritoneum and with fistulas from the pancreas or small intestine It results in numbness and tingling of nose ears tips of fingers and toes muscular and abdominal cramps exaggerated tendon reflexes Electrocardiograms show a prolonged Q-T interval Calcium requires vitamin D for activation

**Calcium excess** (hypercalcemia) may be caused by excessive intake of calcium and vitamin D It is also caused by a number of other conditions such as hyperparathyroidism multiple myeloma Boeck's sarcoid and metastatic cancer It results in osseous manifestations and in calcific deposits in arteries and muscles and at times in the biliary or renal systems It often causes persistent epigastric pains nausea vomiting and occasionally constipation or diarrhea

**Magnesium deficiency and excess** are rarely recognized clinically but are a part of calcium and phosphorus imbalance A deficit will cause symptoms similar to those of hypocalcemia

**Chlorine** an extracellular electrolyte bound both to sodium and potassium plays an important role in maintaining their equilibrium **Chloride deficiency** (hypochloremia) is an accompaniment of both sodium and potassium deficiency or may be entirely the result of excessive loss of hydrochloric acid by vomiting suction or fistulation Sodium and potassium are excreted in the urine in an attempt to maintain body equilibrium An alkalosis is usually present **Chloride excess** (hyperchloremia) is a part of the excess of these two elements

**Phosphorus** an inorganic phosphate is an integral part of potassium metabolism Its concentration in serum falls after the administration of parenteral glucose It is important in diabetic acidosis

**ACID-BASE BALANCE** The pH of the blood is maintained normally by buffers principally by bicarbonates and phosphates plasma protein and hemoglobin Although acidosis or alkalosis can best be determined by measuring the pH of the blood it is usually determined by measuring the carbon dioxide-combining power The normal is 50 to 60 volumes per cent (23 to 28 mEq per liter) 70 per cent (32 mEq per liter) or over indicates alkalosis and 40 per cent (18 mEq per liter) or under indicates acidosis

**Alkalosis** may be metabolic due to an increase of sodium available to form sodium bicarbonate as in excessive ingestion of sodium or decrease in the amount of chloride which tends to cause excessive secretion of potassium Respiratory alkalosis occurs with hyperventilation It must be suspected in any condition mentioned before as causing hypokalemia

Metabolic acidosis is due to decrease of cations or increase in anions reducing the amount of cation available to form sodium bicarbonate Respiratory acidosis is due to retention of carbon dioxide and increased

tension of carbon dioxide within the body. Any interference with alveolar function will result in acidosis. It must be suspected in any condition mentioned before as causing hyponatremia especially fluid loss from the lower gastrointestinal tract as in diarrhea.

This summary of the causes and symptoms of electrolyte imbalances should be a simple guide to the administration of the various electrolyte solutions now on the market. A deficit of any particular electrolyte is an indication for its use. A knowledge of losses of water and electrolytes permits replacement before symptoms of deficiency develop. And a knowledge of the requirements is useful in maintaining balance during periods of stress. In general it must also be realized that the addition of nutritive elements such as dextrose or invert sugar, amino acids and ethyl alcohol is a valuable adjunct to electrolyte therapy.

### HORMONE THERAPY

The use of hormones to aid digestion was much emphasized in the past. *Pepsin* was and still is much used although it is known that rarely even in achlorhydria is there a failure of pepsin secretion. As a rule the administration of dilute hydrochloric acid alone in doses of 1 to 4 cc (15 minims to 1 dram) one half hour after meals is useful in such a case perhaps only because the acid stimulates pancreatic and intestinal secretion which will carry on protein digestion.

*Rennin* was formerly much used as a milk curdling agent in the form of junket which was thought to make milk more digestible. *Intrinsic factor* derived from pig stomach and duodenum has been used for the treatment of pernicious anemia and is still used in combination with vitamin B<sub>12</sub> and folic acid. *Secretin* obtained from the duodenum is used to stimulate pancreatic secretion principally as a functional test in suspected pancreatic disease. *Cholecystokinin* formed in the duodenum stimulates contraction of the gallbladder. It is not necessary to administer it the feeding of fat being all that is needed to stimulate its secretion. *Pancreatin* is used as a substitute for normal pancreatic secretion in cases of pancreatic disease or removal of the pancreas. It is best prescribed in enteric coated tablets.

### ENDOCRINE THERAPY

This formerly consisted largely in treating endocrine dyscrasias principally of the thyroid, pituitary, pancreas and adrenals which might be the cause of some gastrointestinal symptoms.

#### Thyroid

The constipation accompanying hypothyroidism, the diarrhea and occasional vomiting associated with hyperthyroidism have at times been promptly relieved by treatment of the thyroid dyscrasia.

### Parathyroid

The gastrointestinal symptoms associated with hyperparathyroidism are usually promptly relieved by removal of the gland. Medication is of no value.

### Pituitary

The use of posterior pituitary extract in 0.5- to 1 cc. doses (8 to 15 minims) of surgical Pituitrin intramuscularly to stimulate intestinal peristalsis especially postoperatively has been well established. Its pressor principle Pitressin makes it dangerous to use either the Pituitrin or Pitressin alone in hypertension and arteriosclerosis and any of their complications. Pitressin alone has less effect on the intestinal musculature than the whole Pituitrin.

### Pancreas

The effect of insulin on the secretion of hydrochloric acid has been used as a test of such secretion after gastric resection and vagus nerve resection. Its effect in producing abnormal hunger and its irritating effect on the stomach in peptic ulcer patients must be remembered. It has been used to stimulate appetite and when combined with a high caloric diet to put on weight.

### Adrenals

Epinephrine in doses of 0.6 to 1 cc. of 1:1000 solution parenterally or sublingually, aside from its use in shock, has been successful in checking or entirely eliminating allergic reactions. In angioneurotic edema of the gastrointestinal tract due to allergy with symptoms resembling those of an acute condition of the abdomen a dose of epinephrine will frequently cause a complete subsidence of symptoms.

### Adrenocorticosteroid Therapy

Although not all its effects are clearly understood, this type of therapy has been used to excess for all kinds of chronic ailments. Certainly one of its principal effects is on allergic manifestations. It is almost safe to say that if any condition is promptly relieved by this form of therapy, an allergic cause is to be suspected. The prompt relief of symptoms of ulcerative colitis is a case in point. The fact, however, that bowel perforations have occasionally followed its use with symptoms masked by the hormone makes it dangerous to use it in any ulcerative lesion. This form of therapy discourages wound healing and operation should be avoided if adrenocorticosteroids have recently been administered.

### IMMUNOTHERAPY

Natural active immunity to various infections is being produced in the body all the time. Artificial or induced immunity is accomplished in

various ways. For bacterial infection vaccines and serums have been used. For the production of passive immunity in gastrointestinal infections *immune serums* now have been largely superseded by antibiotics. However, when food poisoning is caused only by the toxins of bacteria as in the case of staphylococcus, gas bacillus and botulinus poisonings and some cases of bacillary dysentery, the antitoxins are of spectacular value. *Bacterial vaccines*, whether autogenous or stock vaccines, were long used in the treatment of what were thought to be chronic specific bacterial infections of the gastrointestinal tract, but were shown to be of no specific value and have also been superseded by the antibiotics or sulfonamides. *Immunizing injections* of vaccines for the prevention of specific bacterial infections, such as those of the salmonella and dysentery groups, are of tremendous value and are required to be administered to persons planning trips to areas where such infections are endemic. Protection against *virus infections* is beginning to be accomplished, the Salk vaccine against poliomyelitis pointing the way. At present practically all known intestinal virus infections are of brief duration, but a great saving in illness and time lost will be effected when immunity can be induced artificially.

*Bacteriophage* has been used with success in some intestinal infections, especially pyocyanus infections, but is rarely used now because it is much easier to use antibiotics.

*Nonspecific treatment* of infections by means of *fever therapy* is also rarely used today.

*Desensitization* to allergens is usually included in a discussion of immunotherapy. Specific desensitization is accomplished by administration of increasing doses of allergens by mouth or parenterally, nonspecific by the use of fever therapy, hormone or histamine injections. These will be discussed in the chapter on Gastrointestinal Allergy (p. 79).

### MEDICINAL THERAPY

It is a good general rule not to prescribe medication unless a diagnosis has been made, so that the medication can be chosen accurately to meet the needs of the patient. It would be ideal if we had available specific drugs for every gastrointestinal disease. Unfortunately, this is not so, and most drugs are used only to ameliorate symptoms. A few of each group will merely be mentioned here, with specific recommendations for their use under each disease, discussed elsewhere.

#### Specific Therapy

Specific therapy, so called, includes the use of several types of drugs. *Amebicides* and other parasiticides, the nearest approach to specificity, are numerous, showing therefore that none is really specific. *Antibiotics*, many of which may show specific effects against certain groups of micro

organisms *in vitro* can hardly be classed as real specific drugs their effects varying so greatly in different patients and in different diseases. Many irritate the bowel or modify the flora causing diarrheas hard to treat. Combinations of synergistic antibiotics are now being recommended. The effects of vitamins and minerals in cases of specific symptoms of deficiency or excess have been included under diet therapy. *Antihistaminics* include a large number of drugs supposed to have a specific effect on allergic reactions but these are also uncertain in their effects especially in gastrointestinal allergy. *Antimalarial* drugs if specific would not include so many kinds among them quinine and its derivatives and pyrimethamine. *Antisymphilitics* formerly consisting of mercury, bismuth, arsenic and iodine have now been superseded by penicillin but none will completely eradicate this scourge.

### Symptomatic Therapy

Symptomatic therapy includes the use of several groups of drugs or combinations of drugs known to relieve certain symptoms. Their number is legion new ones are brought out almost daily. Unfortunately, many which are advertised as being specific for certain diseases cannot support such assertions.

*Antispasmodics* Antispasmodics or anticholinergics as they have been called since the virus has been found to be implicated in producing spastic conditions in the gastrointestinal tract are the most abused drugs. Formerly belladonna and hyoscyamus were generally used later atropine. Hyoscyamine which was found to be more of a hypnotic was reserved for "twilight sleep." Belladonna and atropine for years were in every prescription for gastrointestinal symptoms. Fortunately for the patient they caused distressing visual disturbance and dry throat before more toxic symptoms developed. By x ray study it was demonstrated that they had little if any effect on spasm except in excessive dosage. The newer anticholinergics of the Bithine type really relieve spasm and pain but do not promote healing of peptic ulcer so that patients frequently will have perforation of ulcers without symptoms and therefore suffer delayed operation. These drugs also even in small doses often exhibit untoward symptoms ranging from dilated pupils and dry tongue to inability to urinate. As they have no curative properties all these drugs can be easily dispensed with except perhaps for a few days of temporary relief.

*Sedatives* Sedatives and soporifics are today being used indiscriminately. The successors to chloral and bromides the barbiturates are frequently prescribed for almost any symptoms. Since they are habit forming their sale is now restricted. Barbiturate poisoning occasionally fatal is a real problem. The newer sedatives now called "tranquillizers" or "anaraxics" are being used alone and in combination with barbiturates. The many derivatives of *Rauwolfia serpentina* the various preparations



of chlorpromazine the meprobrates and a constantly increasing list of other expensive drugs are being used not only for psychoses as originally recommended but also for any patient who claims to be nervous. As practically all chronic gastrointestinal invalids are neurotics they are in some cases saturated with these drugs. The warnings regarding untoward reactions or allergic manifestations are hidden away in small type in many of the glowing reports of their successful use. They are particularly undesirable if not dangerous for patients with gastrointestinal symptoms. An increasing number of patients have peptic ulcers develop during Rauwolfia medication. Others show jaundice with findings resembling those of biliary obstruction following chlorpromazine administration. Some have nausea or diarrhea from meprobramate. It is much safer not to use any of these drugs in gastrointestinal cases. In incurable cases such as inoperable cancers these drugs are useful as predecessors of or adjuvants to morphine.

*Analgesics* The best pain killer and narcotic except for anesthetics is opium and its alkaloids morphine and codeine.

*Opium* given by mouth or by rectum does not have the immediate effect of morphine and is more constipating. It is now used by mouth mainly in mixtures for diarrhea and by rectum in suppositories for relief of pain in cancer or other incurable diseases. It then requires increasing doses since it is habit forming. It is best combined with belladonna.

*Morphine* prized for its almost instant relief of pain when given hypodermically is of great value in the relief of biliary and renal colic, severe cramps in diarrhea and for the relief of shock. Many young clinicians are inclined to use too large doses. One eighth or  $\frac{1}{8}$  grain is often effective whereas  $\frac{1}{4}$  grain may cause severe respiratory depression especially in the aged; even  $\frac{1}{6}$  grain has been known to cause death.

Three dangers from the use of morphine are ever present: (1) the danger of giving it without establishing that the patient is not suffering from an acute condition of the abdomen when the masking of symptoms and findings may be of serious import; (2) the danger of giving too large or repeated doses while pain persists a procedure that may well cause poisoning when there is a sudden cessation of pain; (3) the danger of inducing the morphine habit. Many morphine addicts date the onset of their craving to the use of morphine to relieve some trifling pain or for its supposed prophylactic value after operation. It should never be used for chronic pain except in incurable patients. (See page 130.)

The effect of morphine in causing spasm of the sphincter of Oddi often spoken of as a contraindication to its use in biliary colic is easily overcome by oral administration of vegetable oils or cream. With this precaution passage of a stone is facilitated and bile appears in the duodenum as soon as a stone has passed or has returned to the gallbladder.

*Codeine* is not as effective a pain killer as morphine but in fairly mild

pains can be used for considerable periods of time without producing a habit. Doses of 1' to 2 grains are commonly used generally in conjunction with salicylates or barbiturates.

Other opium or morphine derivatives such as Pantopon and Dilaudid and newer drugs such as Demerol, Methadone and other substitutes for morphine are also habit forming and are expensive. For severe acute pain there is nothing like morphine. For chronic intractable pain in incurable patients resort may be had to curare preparations, snake venom and nerve block or even cordotomy.

*Coal tar analgesics* especially aspirin either alone or combined with phenacetin, barbiturates and other analgesic and sedative drugs or with caffeine or amphetamine to overcome depressant effects will often relieve pain in gastrointestinal disease even in mild biliary colic. At times all the patient requires is a couple of aspirin tablets to relieve "terrible pains."

*Placebos* Placebos intelligently used are often of great help.

*Anesthetics* General anesthetics are used almost exclusively for surgical operations. In the interest of safety the employment of an anesthesiologist in all abdominal operations is to be recommended.

*Local anesthetics* mainly used for preparation of the pharynx for esophagoscopy or gastroscopy or for the relief of pain in the anal region may be used as applications in the form of solutions, ointments or suppositories or for infiltration or block anesthesia or for operations in that region. Before the intensive study of general anesthesia which has greatly reduced its risks, block and even local anesthesia was used in performing abdominal operations in aged or debilitated patients for whom general anesthesia was considered too great a risk. They may even now be advantageous.

*Antidepressants* Antidepressants are not as numerous as the foregoing depressants. The amphetamines are now the principal drugs used to overcome depression from any cause and are sometimes combined with sedatives or tranquilizers to prevent their depressing effects. As they produce anorexia they are also used in reducing diets and often cause so much mental disturbance as to call for sedatives.

*Gastric Sedatives and Antacids* Gastric sedatives and antacids much used for their supposed local soothing and antacid effects on the mucosa are ingredients of most proprietary "indigestion" remedies. They include bismuth as subcarbonate or subnitrate, various "gel" preparations of magnesium oxide, hydroxide and trisilicate, aluminum hydroxide or phosphate and other salts and alkalis of various kinds including even sodium bicarbonate. Most of these preparations too cheap and therefore not conducive to large profits are combined by pharmaceutical houses with one or more anticholinergics and sedatives and flavored attractively. None of these preparations are required except perhaps

of chlorpromazine the meprobamates and a constantly increasing list of other expensive drugs are being used not only for psychoses "as originally recommended" but also for any patient who claims to be "nervous." As practically all chronic gastrointestinal invalids are neurotics they are in some cases saturated with these drugs. The warnings regarding untoward reactions or "allergic manifestations" are hidden away in small type in many of the glowing reports of their successful use. They are particularly undesirable if not dangerous for patients with gastrointestinal symptoms. An increasing number of patients have peptic ulcers develop during Rauwolfia medication. Others show jaundice with findings resembling those of *biliary obstruction following chlorpromazine administration*. Some have nausea or diarrhea from meproamate. It is much safer not to use any of these drugs in gastrointestinal cases. In incurable cases such as inoperable cancers these drugs are useful as predecessors of or adjuvants to morphine.

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temporarily since nearly all their supposed effects can be accomplished by selected diets

*Stimulants to Acid Secretion* Though bitters have been used they have had no effect in achylia. Substitution of dilute hydrochloric acid is discussed under Hormone Therapy (p 38)

*Intestinal Sedatives Antiseptics and Antidiarrheal Drugs* These are still being used indiscriminately for any diarrhea. Formerly large doses of bismuth usually combined with opium as a sedative and resorcin or salol as antiseptics were most popular. The old bismuth and opium mixtures are still used in acute diarrheas. A single dose of castor oil is often effective in simple diarrhea. To consolidate the feces cellulose preparations kaolin and various antispasmodics astringents and general sedatives are prescribed. The newer antiseptics including sulfonamides and antibiotics have been tried and frequently found wanting. Most of the broad spectrum antibiotics themselves produce diarrheas often associated with *enterocolitis* sometimes fatal. *These diarrheas may be due to direct chemical irritation to allergic reactions to the drug or to the development of microorganisms resistant to the antibiotic.* The staphylococcus is the most serious of these organisms. The insoluble sulfonamides are the safest drugs to use in preparing for operation or for intestinal antiseptics for any purpose. *No diarrhea should be treated until its cause has been determined.* Too many patients die because the occasional diarrhea of an acute appendicitis or the bloody discharges of rectal cancer have been treated as simple diarrheas. (See discussion of diarrhea page 313) When a diagnosis has been made a diarrhea can often be treated medically by diet alone or may require surgical operation.

*Laxatives* Laxatives often a mild name for the most drastic cathartics are still much in use. They are usually taken habitually by persons impressed with the extensive advertising of the danger of constipation. Proper care of constipation by dietetic and hygienic measures will nearly always obviate the need for laxatives. The principal laxative preparations are as follows

*Indigestible bulk preparations* which substitute cellulose agar psyllium and other seeds and various hydrophilic gums for the natural bulk producing vegetables and fruits may be of temporary value as may the use of *antispasmodics* for supposedly spastic constipation."

The various so called *bile preparations* are of course useless for constipation without the addition of some irritating laxative.

*Saline laxatives* of which sodium phosphate milk of magnesia and magnesium citrate are almost the only survivors useful on rare occasions for a "cleaning out" act as irritants when taken regularly and frequently cause so called mucous colitis.

The old *irritants* cascara (alone or in tablets and elixirs) senna (in compound licorice powder and in various other preparations such as

leaves for chewing) aloes (in the ABS pill and in many other forms) and phenolphthalein (in various forms even candies and chewing gum) are still in general use. Several of these cause pigmentation of the rectal and sigmoid mucosa when taken regularly over long periods of time and have been mentioned as possible carcinogens.

*Pituitary hormone* Pituitrin (not Pitressin) is a valuable stimulant to intestinal peristalsis as is physostigmine. Both are given by hypodermic injection.

Formerly many purges were used but now castor oil remains as the most useful. In doses of 1½ to 2 ounces it is the best drug for completely emptying the colon for barium enema x-ray study and proctoscopy. In 1-ounce doses it is used to clear out the bowel in acute diarrheas but should not be used until appendicitis or other acute diseases have been ruled out. *Drastic cathartics* the most irritating exemplified in croton oil are now practically never used.

*Lubricants* preparations of mineral oil (liquid petrolatum) were formerly very popular. They are not laxatives but simply lubricate the bowel promoting movement of its contents. They have been largely abandoned because of the supposed danger that they may remove fat soluble vitamins from the food preventing their absorption. If mineral oil is used properly not more than one tablespoonful at bedtime it passes through the small intestine at a time when no more food or vitamins remain. Although it has been suggested that mineral oil might act as a carcinogen I have never seen a patient in whom it could be suspected of doing any harm. *Large doses of mineral oil* an ounce or more at a time or even smaller doses given frequently produce a characteristic symptom-complex: there may be abdominal discomfort and a frequent urge to defecate; the stools are usually soft formed small in caliber perhaps smeared with oil; there is a feeling as if evacuation has not been adequate (due to the inability of the lower bowel to propel the slippery content) and there may be a leakage of oil with defecation which later leaves the anal region oily and itchy. All these symptoms soon cease when the oil is discontinued and a normal diet is depended upon for a regular bowel habit. Emulsions with agar are irrational since the small amount of agar is not sufficient to produce bulk. Addition of phenolphthalein or cascara is even more irrational since this mixture causes directly antagonistic effects: irritation from the chemicals and soothing action from the oil. But large doses of the irritant are required for laxation and in consequence the oil cannot soothe.

The new *wetting agents* usually containing dioctyl sodium sulfosuccinate soften the feces by encouraging penetration of water. They are not laxatives but promote normal soft formed stools. They are useful in conditions such as partial bowel obstruction in which fecal impaction is a problem and are a help to aged patients. Mild laxatives combined

with the agent are often necessary to aid in expelling the softened feces

*Enemas* The old *soapsuds enema* is still much used for immediate cleansing of the colon. If expertly administered with pelvis elevated tip of tube inserted 2 to 4 inches and the speed of influx regulated to prevent sudden expulsion 2 quarts can usually be injected with ease. It is not used as much as formerly in preparation for operation or rectal examinations nor postoperatively. In low obstruction not all the fluid can be injected and it may return clear. In marked distention it may produce profuse expulsion of flatus. It is useful to remove worms stunned or killed with vermifuges. *Normal saline enemas* are less effective. If hypertonic they may be too irritating and cause considerable discharge of mucus. Small enemas of *cold water* 3 to 16 ounces administered by means of a bag or a small syringe are useful for patients who have weak rectal muscles with lower rectal impaction as in postpartum patients and in the aged.

*Disposable units* containing sodium phosphate and biphosphate solution in 4½ ounce polyethylene squeeze bottles and flexible tubes are used to empty the left colon rapidly to permit endoscopy or to obtain stool specimens. They are given like retention enemas. The unit is discarded when empty.

*Medicated enemas* containing astringents, parasiticides and other medications are not so much used as formerly. They require slow administration of warm fluid from 1 to 2 pints to promote retention.

*Stimulating or "opening injections"* are occasionally useful in stimulating peristalsis in extreme atony. They usually consist of a pint of water or milk containing one tablespoonful each of salt, molasses and liquid soap to which is added one teaspoonful of turpentine for extra irritation.

*Oil enemas* are used for softening impactions. They must be given with the patient lying on the left side and be retained as long as possible preferably for six to twelve hours. The oil (6 or 8 ounces of olive oil or mineral oil) must be allowed to run in slowly by gravity through a warm funnel and catheter and the catheter should be removed with great care to prevent stimulation of peristalsis. At times equal parts of olive oil and castor oil seem to be advantageous. Oil must be at body temperature. Recently disposable polyethylene units containing about 4 ounces of mineral oil have been made available and are convenient to use. *Hydrogen peroxide* also used for softening is dangerous since it might cause perforation of diverticula. It is not to be recommended.

*Colon irrigations* of water, saline and various medicaments were formerly much used. With two tubes or a double barrelled tube in the rectum one for ingress and one for egress of fluid many gallons of solution were injected. These procedures were exhausting to the patient and have in recent years fallen into disuse. The same result can be achieved by the use of simple enemas.

**Rectal Medications** Gelatin enemas are of value in rectal bleeding. Given with the same precautions as oil enemas they can often be retained for a few hours and are soothing, besides being actively coagulant. The solution 10 per cent of gelatin in water (two envelopes of unflavored gelatin in 5 or 6 ounces of water) must be given slowly and hot at a temperature of 112 to 115° F. through a previously heated funnel and catheter and small rectal tube.

**Powder insufflation** into the rectum by means of a powder blower through an anoscope or proctoscope is a form of local treatment for inflammations or ulcerations. Astringents, antiseptics and local anesthetics can be used.

**Suppositories** made of cocoa butter, gelatin or gums designed to melt at body temperature are used in three ways: (1) for stimulating defecation the simple glycerin suppository; (2) for giving medication to patients who cannot swallow either because of weakness or rectal obstruction in an incurable patient when opium and belladonna suppositories in increasing doses starting at 1 grain of extract of opium and  $\frac{1}{4}$  grain of extract of belladonna are a satisfactory substitute for hypodermic medication; (3) for local treatment of rectal irritation due to various causes or to allay congestion in hemorrhoids when combinations of astringents, antiseptics and decongestants are combined as in the various "hemorrhoidal" suppositories.

Other medicinal agents used for the treatment of cardiac, vascular, renal, pulmonary, pelvic, collagen diseases and others which indirectly affect the gastrointestinal symptoms have not been included in this section.

Mercurials and ammonium chloride used to reduce ascites will be discussed under conditions associated with ascites.

### PHYSICAL THERAPY

In the ordinary sense of the term physical therapy is not of any specific value in the treatment of gastrointestinal conditions. In general abdominal massage, general body massage and exercises, sun baths and exposure to ultraviolet light, sprays, baths and other forms of general treatment often serve a useful purpose in toning up the musculature, promoting appetite and soothing the nervous system. But unless carefully controlled and accompanied by proper dietetic care, such treatments can at times do actual harm, especially if the patient happens to have an acute inflammatory lesion.

#### Local Therapy

Local physical therapy would include the application of heat or cold to the abdominal wall by means of bags, packs, fomentations, diathermy,



or some form of radiant or infra red heater to provide heat or the spraying of a volatile liquid such as ethyl chloride to provide cold. Such external applications have little beneficial effect except to relieve pain to some extent and may actually interfere with skin healing if the patient later requires operation. In general it is better to avoid such treatment.

### Balneotherapy

This has been so widely publicized over the years that the public still feels that the waters obtained at spas have some magical specific value. This is of course not true. The various salts and gases in the natural mineral waters are no better or worse than those which can be given if definite electrolyte imbalance can be demonstrated. The principal beneficial effects obtained from a course of treatments at a spa are that a patient who has been too busy or careless to obey ordinary rules of hygiene is placed in a restful environment, is fed properly (though often too much), is instructed to drink sufficient water and is encouraged to take sufficient exercise and rest, the latter often in a tub of water or mud. All these factors have an excellent psychological as well as physical effect. Nevertheless many patients taking too much laxative mineral water will experience severe bowel irritation, many either overeat or starve and many overexercise or rest too much and come home the worse for the experience.

### Lavages

The esophagus, stomach and intestine can all be washed out by physical means.

The *esophagus* is usually washed out by means of a stomach tube, either the large Ewald tube or one of the small caliber gastric or duodenal tubes passed through the mouth or nose. The tube is used to wash out retained food and saliva above a point of obstruction or spasm or for removal of swallowed poisons. Water or saline solution is usually the medium used, although in some cases alkaline or medicated solutions or antidotes may be indicated. An oil may be left in for its soothing effect.

The *stomach* may also be lavaged through stomach tubes, large or small. Water, saline, alkaline or medicated solutions or solutions containing antidotes to ingested poisons may be used for this purpose. The washing fluid may be introduced through the tube until the stomach is comfortably distended and then removed by suction or siphonage. Washings are repeated until the return is clear. It is sometimes easier to have the patient drink the solution alongside the tube and to remove it by means of the tube. Lavage was formerly a favorite treatment for all kinds of stomach ailments, especially gastritis, but today is used principally for removal of noxious agents swallowed by the patient or to clean

out the stomach once or twice daily in cases of pyloric stenosis. In the latter cases it also stimulates gastric peristalsis.

*Gastric autolavage* is accomplished by having the patient swallow rapidly a pint or more of water or hypertonic saline solution (two tea spoonfuls of sodium chloride to a pint of water) and then immediately encouraging or inducing vomiting. It is a useful method of removing non corrosive poisons such as opiates or barbiturates when no stomach tube is at hand. It is of great value in vomiting due to pylorospasm or retrostalsis as in postoperative ileus and in vomiting of pregnancy. In the latter cases a meal given within fifteen minutes after the procedure will often stay down. If not the whole procedure is repeated. If it is not successful after two or three attempts real obstruction must be suspected.

*Transduodenal lavage* consists first in passing a duodenal tube making sure by x ray examination or by aspiration that it is in the duodenal loop. Then one pint of a warm hypertonic solution such as 5 per cent each of magnesium and sodium sulfate is slowly run into the duodenum and the tube withdrawn. In the absence of obstruction within twenty minutes there occurs a profuse watery catharsis cleaning out the entire intestinal canal. Such lavage is particularly useful for the prompt removal of poison administered to kill or anesthetize worms or other intestinal parasites and to remove the parasites themselves.

### Enemas

Instillation of a solution into the rectum for the treatment of disease is one of the oldest forms of physical therapy. Injection of cold water or actual ice water has been used for the reduction of fever and injection of hot liquids in the treatment of hypothermia as in shock. Various kinds of rectal injections have been used to empty the bowel in constipation or to soothe it in diarrhea. Nutrients and medications are still given by rectum. The subject of enemas has been discussed in the section on Medicinal Therapy (p. 46).

### Electrotherapy

Galvanic and faradic currents have been used to stimulate peristalsis but are of doubtful value. Heat therapy has been mentioned above.

*Fulguration or electrodesiccation* popularly called the "electric needle" is used for removal of benign and malignant growths from the rectum and at times from the esophagus. Benign tumors can be neatly removed. Carcinoma of the rectum has been successfully removed by this method the extreme heat apparently having an effect on any neighboring tissue involvement. It has been used mainly in advanced cases and even in these three or four desiccations will at times succeed in removing the growth and if no distant metastases have occurred will result in cure.

### Radiotherapy

This includes x ray radium and radioisotope (principally cobalt and gold) therapy the use of which should be left to radiologists who are expert in this field. This form of therapy is used principally in the treatment of cancer. Many of the lymphomas are susceptible to radiation and may be at least reduced in size or held in check by this form of therapy combined with chemotherapy. Some especially Hodgkins disease are not curable but may be so controlled that life may be prolonged for years during the treatments.

*Carcinoma* of the gastrointestinal tract is probably never cured by radiotherapy alone. The damaging effect on the tissues especially the liver of the large doses required for destruction of cancer makes this treatment only palliative. The only location in which radiation will have a good effect is in the rectum. But even here the safest method for its use is to reduce the growth to a point at which surgical extirpation can be accomplished.

*Prophylaxis* For the purpose of destroying metastases which may have occurred without showing evidences of their presence radiotherapy has been used both before and after operation. There is still considerable controversy as to the value of this procedure.

*For Inflammation* The favorable effect of radiation upon early inflammatory reactions and upon exudates warrants its occasional trial in colonic diverticulitis. Inflammatory masses suspected of being malignant will at times subside quickly after one or two mild treatments.

*Caution* The amount of radiation given to a patient even under an average fluoroscopic study and especially in the course of a prolonged dental x ray study at times exerts an unfavorable effect upon the patient's gonads and various congenital anomalies due to the effect of radiation of the genes may be carried through generations thereafter. It is therefore wise to avoid excessive exposures and to protect the gonads carefully by means of lead plaques or aprons.

The effect of radiation in opening up avenues for the spread of infection and the harmful effect on the skin often preventing wound healing are additional reasons for avoiding the indiscriminate use of this modality.

### ONCOLYTIC THERAPY

All over the world research is being conducted in the hope of finding some agent which when administered orally or parenterally will dissolve all tumor tissue in the body. Various agents have been produced which were extensively publicized as cancer cures and have been found wanting. The latest of these appears to be Krebsiozen which still has important backing. Hormones especially gonadal extracts and corticosteroids show some effects in controlling growth of cancer. Eschrotics have been used

externally but are not safe to use in the gastrointestinal tract. Experiments with radioactive gold and other radioisotopes are showing some progress. Mercaptopurine and azocytidine have shown limited effects. Possibly if cancer is due to a virus as some scientists have suggested we may look for an oncolytic agent in the future. It would be the only real cure for cancer.

### PSYCHOTHERAPY

Psychotherapy an important part of the treatment of gastrointestinal diseases is discussed in the chapter on Psychosomatic Disturbances (see p. 131).

### SURGICAL THERAPY

The great improvement in surgical technique, the increasing safety of anesthesia and the successful prevention and treatment of operative and postoperative complications have resulted in relief and in many cases cure of conditions formerly considered inoperable and incurable. Unfortunately the knowledge of these factors has been a spur to much needless surgery, especially by incompetent operators. General practitioners and internists are being consulted by increasing numbers of patients suffering from the complications of surgery often performed without justification. Many of these unfortunate patients undergo repeated operations for these complications. Because of the many extensive articles in lay publications describing the brilliant results from surgery patients are demanding operations for all kinds of abdominal symptoms and keep "shopping" until they find someone who will operate.

It is important to recognize the indications for and contraindications to operations upon the gastrointestinal tract, to know what operation can accomplish and what complications may occur after operation.

#### Contraindications to Operation

Contraindications in general would include old age, advanced cardiovascular renal disease, diabetes, marked debility, including nutritional deficiency and dehydration, and the presence of an acute infection, especially of the upper respiratory tract. None of these are today considered absolute contraindications. By proper choice of anesthesia administered by an expert anesthesiologist, even extensive operations on the aged are being carried out successfully. Patients with diabetes, cardiovascular and renal diseases can safely be operated upon under the guidance of experts in these fields. Nutritional and electrolyte disturbances and anemia can be corrected, acute infections eliminated. In acute emergencies requiring immediate operation many of these treatments can be successfully carried out during the short time before operation, during operation and afterwards.

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tine and other organs such as the bladder the vagina or the skin are also complications requiring operation often in two or more stages

*Neoplasms or Suspected Neoplasms* Removal of *benign tumors* is indicated to relieve obstruction and to prevent possible malignant degeneration Persistent thickening and marked true polypoid changes in the bowel wall frequently require operation especially because they may be due to malignant changes or may become malignant

*Cancer* is treated surgically in two ways

**COMPLETE EXTIRPATION** Until some other cure for cancer is discovered complete removal of the growth will remain the only hope of its cure In cases in which it is uncertain whether such complete removal can be accomplished it is nevertheless worth while to attempt it since at times cures are effected even when extensive growths are removed Recent studies seem to indicate that the wide resections often of much normal tissue and of uninvolved distant lymph nodes are not only unnecessary to effect a cure but also may actually promote metastases by removal of the protective barrier of lymph nodes

**PALLIATION** Though not curative palliative operations will often make a patient more comfortable and may even prolong life They may consist in local excision of the growth in anastomoses bypassing the lesion or in providing an external opening in the skin The indications for each are discussed in the chapter on Gastrointestinal Cancer (p 126) and under each specific organ

*Exploration* Except in the case of an acute condition when it is definitely indicated exploratory operation indicates a failure of diagnosis It should never be done except in cases in which after a complete study there is uncertainty about the diagnosis especially if malignancy is suspected Of course in a general sense all operations are exploratory and even though a lesion is found that definitely requires operation a careful exploration of the entire abdomen and pelvis should be performed so that any other lesion can be cured for at that time or on some later occasion A long scar is usually a sign of a good exploration

### Complications of Operations

During operations complications of two types may occur First are those due to the stress of operation and effects of the anesthesia These include not only such direct effects as shock asphyxia and toxic effects of the anesthetic agent but also effects on the heart vascular apparatus brain and lungs Avoidance of these complications and treatment if they occur are best accomplished by an experienced anesthesiologist who today is in charge of the patient's medical condition while the surgeon operates When such an expert is not available a competent clinician should be present to cope with emergencies Parenteral fluids electro

Barring an emergency however it is better in patients with these contraindications to postpone operation if possible until they have been brought to a point of optimum operability. This is true even of patients in fairly good general health. Except in an acute abdominal calamity a candidate for operation should be properly fed, should receive fluids, vitamins and minerals, should avoid stresses and strains and should have all even minor ailments carefully treated for at least two weeks and preferably longer before operation.

An important part of the preparation for an abdominal operation because it will prevent postoperative complications should be the elimination of upper respiratory infections, removal of dead teeth and infected tonsils and cleaning up of infections elsewhere. Before operation upon the colon sterilization of the bowel by antibiotics or better insoluble sulfonamides was often done but is now being frowned upon. With adequate precautions surgery will produce the results hoped for when any patient is advised to have an operation. Details of preoperative and postoperative care are discussed under each disease.

### Indications for Operation

Indications for operations upon the gastrointestinal tract can be divided into five groups each done for a definite purpose.

*Emergencies* as described in the section on Acute Abdominal Condition (p 59). When an acute condition is suspected no analgesics should be administered until after consultation with a surgeon. Other preoperative care is however very important.

*Relief of Mechanical Disturbances* This would include cicatrizations, kinks, adhesions, deformities and partial obstructions, whether congenital or acquired from disease or operation. Great care must be exercised in careful selection of cases for operation, using every diagnostic means to determine the exact nature and cause of the lesion and a period of trial of medical care before deciding on surgery. *Operations for adhesions are often unsuccessful because new adhesions tend to occur.*

*Complications* Though mechanical factors such as those just mentioned are frequently complications of gastrointestinal lesions, the most frequent complications requiring elective and not emergency operations are *walled off perforations* of peptic ulcer, intestinal and colonic ulcers and colonic diverticula. These perforations form accessory pockets whose base, not being the gastrointestinal wall, shows little or no tendency to fill in or become obliterated. It is usually desirable that such pockets be excised and suitable anastomoses done to restore gastrointestinal continuity. *Calculi* in the gallbladder and occasionally in the pancreas usually call for surgery. *Fistulas* between gallbladder and stomach or intestine between different parts of the intestine and between the intes-

tine and other organs such as the bladder the vagina or the skin are also complications requiring operation often in two or more stages

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lytes blood and various stimulants may be required Cardiac massage by the surgeon may be called for

Second are complications occurring in the operative field These include hemorrhage leakage from perforations accidental ligation of the common duct ureter or essential blood vessels failure to remove all gall bladder calculi or all the gallbladder overlooking removal of instruments or sponges from the abdominal cavity and others The surgeon and his assistant must be constantly on the lookout for these conditions so that they can be corrected before the abdomen is closed

After operations complications preventing an uneventful convalescence would include all those mentioned as occurring during operation and continuing their effects afterward as well as ileus infection failure of wound healing peritonitis and acute exacerbations of previous chronic lesions such as pancreatitis ulcerative colitis diverticulitis and peptic ulcer

Late complications may be due to a continuance of symptoms of any of the complications just mentioned They may consist of backaches and other aches and pains resulting from the posture on the operating table injuries from spinal anesthesia and functional disturbances resulting from inadequate postoperative dietary and general care Hernias fistulas adhesions angulations and conditions overlooked at operation may require further operative procedures later Besides this development of keloid or even at times of cancer in a scar may be a surgical complication

### Postoperative Care

Prevention of postoperative complications is most important Aside from the prevention or treatment of shock by parenteral administration of fluids electrolytes alcohol blood and any indicated cardiac stimulants the cardinal points in such a program consist in early feeding and early ambulation

*Feeding* Parenteral feedings such as those described before (p 35) are of value but oral or tube feedings begun even on the day of operation are essential to stimulate gastrointestinal secretion and motility and to prevent postoperative ileus They are better even than the much overdone routine continuous suction and provide the nutrition needed for wound healing and general recovery from the operation They are discussed under the various diseases requiring operation

*Ambulation* Ambulation is now started on the second day after most operations and is preferably preceded by exercises in bed Deep breathing aided by carbon dioxide and oxygen inhalations or the "blow bottle" will usually prevent pulmonary complications Exercise and walking promote good general circulation prevent phlebitis keep the muscles in

good condition and contribute to a feeling of well being and confidence in recovery

*Excretions* Urination must be watched bladder distention promptly treated and the heart and lungs must be examined frequently *Defecation* is usually not important With early adequate feeding the bowels may move spontaneously on the third or fourth day Retention oil enemas may be of use Enemas and cathartics very exhausting, to some patients should be avoided if possible The treatment of ileus is discussed elsewhere (p 310)

*Psychologic Care* This is a great help to the patient Except in cancer early explanation of what was found and the reasons for the operative procedures carried out will inspire the patient with confidence and hope and encourage his cooperation in whatever measures are being used

### Prognosis

Prognosis in regard to operation should always be guarded Even though the surgeon reports only a 1 or 2 per cent mortality for a given operation there is no assurance that a given patient will not be one of these fatalities It is also evident from a study of the partial list of complications just mentioned that many patients though surviving the operation will have much trouble afterwards It is therefore essential not to advise operation for any patient except in emergency until it is reasonably certain that the operation will benefit the patient instead of leaving him a gastrointestinal invalid

### Written Report

A written report of the operative findings and procedures should be sent to a referring physician and unless it contains information which a patient should not know about a copy of the report should be given to the patient so that it can be shown to doctors whom the patient may consult in the future Many surgeons now do this

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## SECTION II

# Diseases Affecting the Entire Gastrointestinal Tract

It is expedient to consider in one section the diseases affecting the entire length of the gastrointestinal tract even though some may affect different parts of the tract in various ways and with differing degrees of severity. Some poisonings, infections, infestations, allergic manifestations, cancer and even certain developmental defects while affecting one region primarily will usually cause symptoms and perhaps actual involvement of other parts or the whole of the gastrointestinal tract. The most important ones will be discussed together in this section with local manifestations described in the chapters devoted to different organs of the gastrointestinal tract.



# Acute Conditions of the Abdomen

The term "acute abdomen" "acute surgical abdomen" and "acute abdominal calamity" include acute diseases due to five pathological conditions: acute perforation, infection, hemorrhage, obstruction, and vascular occlusion. All these conditions are of the gravest significance, are usually associated with more or less acute peritonitis, and all are frequent causes of death if not recognized early. Whereas any one of these conditions may be present alone at the onset, most cases exhibit evidences of one or more of the others soon thereafter. For instance, an intestinal obstruction may cause vascular occlusion, hemorrhage, and peritonitis; an acute appendicitis, perforation, obstruction, hemorrhage, and vascular occlusion; an acute hemorrhagic pancreatitis, peritonitis; a mesenteric thrombosis, obstruction, hemorrhage, perforation, and peritonitis. An acute condition must be suspected in any case of trauma. The differential diagnosis in advanced cases is therefore difficult, and the most that can be determined is that a calamity has occurred and that operation is urgently indicated.

Operative mortality in such cases is extremely high. It is of the utmost importance for the first doctor who sees the patient in the early stages to recognize the gravity of the situation, to avoid injuring the patient by unnecessary or harmful medications or procedures, and to call for prompt surgical consultation. Too many patients have died because the first doctor was careless, the most frequent offenses being the prescription of a laxative, an enema, an antibiotic, or analgesics.

## Mimicry

The fact that so many nonsurgical, even nonabdominal, conditions may mimic an acute surgical condition of the abdomen makes it imperative early, by careful history and physical examination, to rule out such conditions. Many patients are erroneously subjected to abdominal exploration without benefit or with actual harm when the real condition was a pneumonia, especially at the right base; myocardial infarction or congestive heart failure; hemoperitoneum during anticoagulant therapy; food or other poisoning (acute gastroenteritis); acute hepatitis; herpes zoster; and other spinal lesions, neuritis, myositis, hysteria, especially with excessive aerophagia; mesenteric adenitis; collagen disease; and a variety of other conditions, including dissecting or ruptured aneurysm, diabetes, acute childhood diseases, mesenteric adenitis occurring with an acute upper respiratory tract infection, diabetes, sickle cell anemia, lead poisoning, and porphyria.



the pain is intense. They may also not occur in colonic obstruction in spite of distention. If pain is not relieved by vomiting it suggests an organic cause.

The *type* of the vomiting must be noted. *Projectile* vomiting, although usually indicative of a central nervous system lesion, may also occur but with severe nausea in acute pancreatitis. *Forceful* vomiting usually occurs in any acute abdominal lesion. The *welling up* of gastric contents without force or effort occurs in advanced pyloric stenosis but is also present in any severe prolonged neglected acute abdominal condition.

The *quantity* of vomitus is greatest in obstruction of the jejunum and upper ileum and consists of ingested material as well as of the secretions of salivary, gastric and duodenal glands, bile and pancreatic juice which have not had a chance to be absorbed. Lower obstructions do not produce such voluminous vomitus.

The *appearance* of the vomitus helps in determining its origin. The presence of bile shows that there is no biliary obstruction. Recently ingested food not yet digested indicates retrostalsis from obstruction or inflammation fairly high up in the intestine but food taken ten hours or more previously indicates pyloric or upper duodenal stenosis. Blood usually indicates ulceration or sloughing.

The *odor* is usually merely sour or may partake of the odor of ingesta. In prolonged stasis due to obstruction at any point but usually proximal to the cecum bacterial activity will produce a fecal odor and the content will resemble liquid feces.

### Other Symptoms

Other symptoms not as constant include the following.

*Abdominal distention* may or may not be present and is often a sign of prolonged obstruction or of peritonitis with paralysis of intestinal musculature. *Borborygmi* are prominent in obstruction accentuated at the time of the pain. They are usually absent in peritonitis.

*Feter* may be absent. Often if present it indicates peritoneal irritation or infection and is evidence also of toxic absorption.

*Defecation* may be influenced. Early in obstruction there may be normal stools later *obstipation* which depends upon the location of the obstruction. It is also usually present in peritonitis. *Diarrhea* may be an early symptom, may be present in retrocecal appendicitis even with perforation and is often present but scanty with blood in intussusception and in rectal cancer.

### PHYSICAL EXAMINATION

The examination may be of great value or may be *deceptive*. The patient may have the physical findings of an upper respiratory tract



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An important condition resembling an acute abdominal lesion is an acute allergic reaction occurring in some part of the gastrointestinal tract. This is discussed in the chapter on Gastrointestinal Allergy (p. 79). To rule out allergy a single intramuscular injection of 10 or 15 minims (0.6 to 1 cc.) of 1:1000 epinephrine solution can be tried with out harm to the patient. In an allergic reaction it often produces dramatic relief of symptoms. *Ephedrine is much less effective.*

Although all of the above mentioned conditions may closely simulate an acute abdominal condition it must be borne in mind that each of them may be complicated by a real abdominal calamity and should not be treated without a careful examination to rule out the others.

*Adynamic (paralytic) ileus* is another confusing condition in which usually the entire gastrointestinal tract is greatly distended in most cases with swallowed air. The aerophagia is caused by the patient's efforts to bring up what is thought to be gas. Ileus may be the result of any form of true abdominal calamity but it may also be caused by a great variety of nonsurgical conditions in fact any of those just mentioned as resembling an acute abdominal lesion. It may also result from reflex inhibition of intestinal peristalsis as a result of surgical operations, prolonged anesthesia, fractured ribs, abdominal and central nervous system injury, hyperextension of the body in a body cast, acute fevers and sepsis. In the nonsurgical lesion the distention usually involves the entire tract, peristalsis is absent or slight and pain is usually a minor symptom. The measures short of operation which are used in the treatment of intestinal obstruction will usually relieve this condition.

### SYMPTOMS

The cardinal symptoms of an acute condition of the abdomen are pain, nausea and vomiting. One outstanding feature is the general reaction to these symptoms of the patient who is usually more or less prostrated, complains of thirst and of feeling very ill in general and insists that something must be done about it promptly.

#### Pain

The pain usually comes on suddenly, is usually severe, may be constant or intermittent and regardless of the location of the lesion may start in the epigastrium or mid abdomen and thereafter become localized at the site of trouble. Even in early obstruction cramplike pains lasting only a few minutes occur periodically, owing to an effort on the part of the intestine to push contents through the narrowed area.

#### Nausea and Vomiting

Nausea and vomiting may accompany the pain early or late except that in perforation of a peptic ulcer they are notably absent although

*Borborygmi* the noises accompanying peristalsis are a sign of intestinal peristalsis. It is worth while to listen to them through a stethoscope. If they occur periodically and are high pitched with their maximum associated with pain they are highly suggestive of intestinal obstruction. They can also occur however with acute food poisoning or enterocolitis from any cause.

*Ascites* appears late in intestinal obstruction and to some degree in a free perforation and peritonitis. It may be caused by transudation, exudation or hemorrhage. Examination of ascitic fluid may give valuable information. A small amount may be safely removed with an 18 or 20-gauge needle with the patient in a semi sitting position.

### Rectal Examination

Rectal examination should not be neglected. The finger may feel a rectal cancer or an intussusception whereas high up in the cul de sac a Krukenberg tumor or peritoneal fluid may be detected. In an ectopic gestation and with pelvic abscess tenderness will be felt and often a mass. A low lying inflamed appendix will show right pelvic tenderness.

### Late Findings

In the final stages of an acute abdominal condition there is severe *dehydration* and the patient goes into profound *shock*. Evidences of venous stasis occur and the abdomen becomes definitely distended.

## LABORATORY EXAMINATION

### Blood Examinations

*Leukocytosis* with increased polymorphonuclear leukocytes while often a sign of acute infection and if excessively high of perforation may also be due to any intercurrent infection such as pneumonia and even an acute upper respiratory tract infection. A normal blood cell count may be present in the early stages of an acute condition of the abdomen and may never become high throughout. A high *sedimentation rate* may also prove deceptive. In sudden hemorrhage *anemia* may not be shown until later unless blood volume is estimated. Blood *chemistry* studies will in general not be of great help although elevated amylase and calcium values occur with acute pancreatitis, low calcium and high sodium levels in high small intestinal obstruction.

### Urine Examination

Urine examination is of value principally as a check on the kidneys. Glycosuria which has not existed previously suggests pancreatic disease.

infection of pneumonia or pleurisy of a cardiac lesion a pelvic disease or any of the conditions aforementioned as *mimicking* an acute abdominal condition and yet have coincidentally the signs of an acute calamity. *Restriction of movement of the diaphragm and evidences of fluid in the pleural cavity occur with peritonitis high up* as from perforated peptic ulcer and acute pancreatitis. A careful complete physical examination is important but the abdominal examination requires the greatest care.

Thorough search for a *hernia* must always be made with the patient in the erect recumbent and stooping positions not only in the inguinal and femoral regions but also in the umbilicus in operative scars and in the epigastrium. Incarcerated and strangulated hernias are frequently overlooked especially when the patient is lying down. Inquiry should always be made about the existence of a hernia.

### Abdominal Examination

The principal findings on abdominal examination are as follows.

*Tenderness* is important especially if localized and severe. The degree of tenderness is difficult to evaluate especially in a hysterical patient. In general it may be said that the patient usually cannot be distracted so as to disguise a real tenderness. Localization in the area of the lesion usually occurs eventually. If over a mass it is particularly important. Rebound tenderness is a valuable sign of peritoneal involvement. Right flank tenderness is suggestive of a retrocecal appendix.

*Rigidity* is usually present with peritoneal irritation. It may vary in degree from muscle guarding to spasm to real boardlike rigidity. It may occur first over the lesion spread to the entire rectus muscle on that side and finally with general peritonitis will involve the whole abdominal wall. In late stages flaccidity ensues and there remains only some soreness.

A *mass* usually occurs late and is due to abscess formation following perforation or to a cancer an intussusception or volvulus.

*Abdominal distention* or ileus due mostly to swallowed air may occur in hysteria but persistent distention is a bad sign. It is important to recognize the pattern of the distended intestine by both inspection and percussion. Distention in the mid abdomen due to a dilated small intestine is usually caused by obstruction near the ileocecal region. Definite right sided distention of the cecum ascending colon and transverse colon as far as Cannon's ring is a valuable early sign of obstruction in the pelvic colon. Distention of the whole colon is a late sign of colonic obstruction. Absence of liver dullness over the lower ribs is often found with perforation of a hollow viscus but liver ptosis must be ruled out. At times *auscultation* may elicit faint bubbling sounds over the site of a perforation.

pelvic colon. General colonic distention may be of no significance or may indicate late paralysis of colonic musculature (Fig 7). It may be entirely due to aerophagia (see Fig 2 p 9). Demonstration of ascites is a grave sign.



Fig 4



Fig 5

*Figure 4* Free air in peritoneal cavity. Erect film. (1) liver pushed down by (2) air under the diaphragm. (3) air under the left diaphragm. (4) gas in the descending colon.

*Figure 5* Free air in peritoneal cavity. Lateral recumbent film. (1) (2) air at highest point. (3) gas in colon.



a



b

*Figure 6* a Jejunol obstruction. Miller Abbott tube in duodenum. (1) stomach filled with air. Below this the ladder-like arrangement of distended small intestinal coils extends to the pubes.

b Small intestinal obstruction from artery clamp left in at previous operation. Miller Abbott tube has not gone through the pylorus. Ladder-like arrangement of distended small intestinal coil. No gas in colon.

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Hematuria may be of importance but is not definitely diagnostic even in a catheterized specimen. The finding of calculi or of crystals with the blood would suggest that the patient's pain came from their passage.

### Gastric Analysis

In a suspected acute condition gastric analysis should not be performed although examination of specimens obtained from the stomach and duodenum on introduction of a suction tube may give information regarding bleeding or may contain cancer cells. Examination of vomitus is important. It may consist only of normal gastric and duodenal content with or without food, may show blood or may resemble liquid feces.

### Paracentesis

*Ascitic fluid* may give a valuable clue to diagnosis. Whether it is a transudate or exudate should be determined. blood, bile and fat should be looked for. *Cell block* may disclose cancer cells. In acute pancreatitis amylase may be found for a few days after it has returned to normal in the blood.

### Stool Examinations

Visible blood suggests cancer or polyps, intussusception or gangrene of obstructed gut. Occult blood is of no importance. The acholic stools of common duct obstruction may precede gallstone ileus. Fatty stools may also occur with biliary obstruction or pancreatic disease. *Cytologic studies may disclose cancer.*

## X-RAY EXAMINATION

Scout films can usually be safely taken if the patient is in a hospital. In a suspected case the scout films should be taken upon admission before the patient is sent to his bed. The following findings are important.

*Perforation* of a hollow viscus may be demonstrated in films taken in the supine, lateral and erect or semi-recumbent positions by the finding of shifting gas or air appearing at the topmost areas—subdiaphragmatic and suprahepatic or in the flanks (Figs. 4, 5). In perforation following intestinal obstruction the gas may be seen along the lateral margin of the liver.

*Intestinal obstruction* can be beautifully demonstrated. The mid-abdominal ladder-like arrangement of the coils of the small intestine in ileocecal obstruction is characteristic (Fig. 6). In some cases isolated loops of intestine may be seen. In jejunal obstruction distention is not always prominent because of frequent vomiting. The dilatation of the proximal colon to Cannon's ring is indicative of early obstruction in the

allergic manifestations being of help in arriving at this diagnosis. In a female with attacks occurring halfway between menstrual periods ovulatory bleeding may be suspected but such attacks usually subside in twelve hours.

Although many times an exact diagnosis cannot be made and the patient must be subjected to operation with a diagnosis merely of "acute abdomen" there are some findings which may be of help in differentiating the possible causes of this calamity. Following is a brief résumé.

### Intestinal Obstruction

Intestinal obstruction occurs most frequently as a result of external hernias and internal adhesions and bands. A history of hernia and of previous operations especially pelvic operations or intestinal anastomoses is therefore invaluable. A recent biliary colic or suddenly subsiding jaundice suggests gallstone ileus due to ileocecal valve obstruction by a gallstone which perforated into the duodenum. A history of chronic gastrointestinal symptoms with bleeding and loss of weight for a few months before obstructive symptoms suggests malignancy which may cause obstruction or perforation. The history together with a thorough physical examination and if possible the laboratory tests mentioned must be thoroughly digested by the group of consultants. It is risky but at times justifiable in rare mild uncertain cases to postpone decision and to hold a further meeting in four to six hours meanwhile leaving a competent observer with the patient to detect untoward signs which might make immediate operation imperative.

Such signs would include (1) sudden increase in pain rigidity and rebound tenderness (with or without fever and leukocytosis) indicating peritoneal involvement (2) steady increase in colicky pain distention and vomiting increasing evidences of toxemia rapid weak pulse lowered blood pressure cold extremities sweating, and all evidences of shock together with increasing tenderness and rigidity and perhaps evidence by palpation of a mass indicating strangulation (3) Sudden cessation of pain may follow the excruciating pain of a perforation succeeded later by signs of peritonitis (4) Failure of pain and other symptoms to subside after several hours even without direct evidence of either peritonitis or strangulation indicates that surgery will undoubtedly be required.

Although the typical findings described above will often establish the diagnosis of obstruction and later strangulation at times it is difficult to differentiate it from an acute inflammation or perforation with peritonitis. This is especially true when strangulation has resulted in peritonitis. As strangulation often occurs with the onset of an obstruction this adds further confusion. Marked dilatation of the stomach and intestine following any operative procedure with all the symptoms of ileus



Figure 7 Colonic obstruction. The ascending, transverse and descending colon is greatly distended. Barium enema shows obstruction at rectal cancer (1)

*Barium by mouth* is definitely contraindicated in any acute gastrointestinal condition.

*Barium enema* study may be done with great care when a colonic lesion or obstruction is suspected if the patient is not too prostrated.

#### DIAGNOSIS

The decision as to whether a patient is suffering from an acute abdominal lesion and whether early operation is indicated is often difficult and of serious import. It requires the best judgment, keen surgical appreciation and long experience to arrive at a diagnosis. It is always advisable to have early consultation with a surgeon and preferably also with an internist. There is often insufficient time and equipment to afford extensive laboratory studies and even if these are available the findings cannot be accepted unequivocally since they may often be misleading.

Speed is important but it is unwise to rush the patient into the operating room until a careful history has been taken. A patient with a history of similar attacks which have subsided rapidly may have had temporary intestinal or appendiceal obstruction. He may have had recurrent attacks of appendicitis, cholecystitis, pancreatitis, regional enteritis or colonic diverticulitis. He may have had allergic reactions, a history of other

allergic manifestations being of help in arriving at this diagnosis. In a female with attacks occurring halfway between menstrual periods ovulatory bleeding may be suspected but such attacks usually subside in twelve hours.

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is a serious challenge to diagnosis. Differentiation between mere atony, an obstruction, a hemorrhage or a perforation is often possible only at operation or autopsy.

The most serious factors in obstruction are the loss of fluids and electrolytes, toxic absorption and interference with circulation, causing strangulation, hemorrhage and peritonitis. Fifty per cent of deaths are caused by shock and peritonitis.

### Acute Appendicitis

The persistently high mortality rate from *acute* appendicitis has been shown to be due to neglect as a result of mistaken diagnosis so that perforation, peritonitis and abscess formation have been encountered at operation instead of a simple catarrhal appendicitis. In a typical case the diagnosis can be made by telephone and usually even the patient makes his own diagnosis. About one fourth to one third of cases, however, do not show the typical findings.

At times *crampy* pain may be the only symptom with few physical signs of disease. Patients *without* pain, usually the elderly, show the highest death rate. In some cases, usually dependent on the location of the appendix, the pain may be in the lumbar region (in the retrocecal appendix), in the gallbladder area or in the right shoulder.

X-ray scout films showing bubbles of gas next to the cecum or over the right iliac wing may be found in perforation of the appendix; gas in the right flank can be seen in perforation of an appendiceal abscess.

*Nausea* and *vomiting* may be the only symptoms or may be absent.

*Atypical Findings.* Some patients complain only of malaise, vertigo, faintness, headache or backache, some of urinary symptoms, some of indigestion, of diarrhea, of hiccup or anorexia. Some have no fever, others have chills and fever, and few if any have abdominal symptoms. Hematemesis, melena and epistaxis may be the most prominent manifestations. Onset with diarrhea may simulate acute gastroenteritis or acute regional enteritis.

*Abdominal Examination.* In acute appendicitis, even with peritonitis, abdominal examination may show no tenderness, rigidity or distention, especially when the appendix is retrocecal. Tenderness may be entirely pelvic and lumbar or subcostal. Pulse rate and temperature and the blood cell count may be normal until later when infection has occurred. Obesity may mask all physical findings.

*Errors in Treatment.* In recent years many patients have lost their lives because the symptoms were obscured or altered by *antibiotic therapy*. Abscess formation and peritonitis may occur during such therapy. In spite of warnings to doctors and to the public to avoid *cathartics*, *analgesics* and *sedatives* until an abdominal pain has been

carefully studied patients are regularly being admitted to hospital with perforation and peritonitis as a result of such misguided therapy. Early operation is the only safe treatment.

### Other Causes of an Acute Condition in the Abdomen

*Regional enteritis* may cause symptoms similar to those of appendicitis and may be associated with it. Careful examination of the ileum at operation is always imperative.

Inflammation or perforation of a *Meckel's diverticulum* will also cause identical symptoms. At operation an inexperienced surgeon may remove a questionable appendix and miss such a diverticulitis with grave consequences.

*Colonic diverticulitis* usually occurring in the sigmoid gives symptoms like those of appendicitis but on the left side. A history of the previous finding of diverticula may aid in diagnosis but may also confuse in that a true appendicitis may have left sided symptoms.

*Perforation of a peptic ulcer* although usually causing epigastric pain and prompt severe tenderness and rigidity the patient lying perfectly still and terror stricken may show atypical symptoms. The sudden cessation of pain followed by signs of peritonitis has been mentioned. A small perforation may be promptly walled off by adhesions to the pancreas liver or omentum with fairly early relief of the severe symptoms. At other times it may allow gastric contents to migrate upward above the liver producing a subdiaphragmatic abscess. Perforation into the pancreas often produces symptoms of acute pancreatic necrosis. It is not safe to assume that a patient has such an acute pancreatitis and treat him expectantly since the adhesions may separate and general peritonitis ensue. The fact however that many so called chronic ulcers are in reality pockets formed by such walled-off perforated ulcers makes it evident that perforations can occur without violent symptoms.

*Acute pancreatitis* is associated with equally severe pain often referred to the back but usually with accompanying nausea and projectile vomiting distinct tenderness distention tachycardia cyanosis and an abdomen of doughy consistency. Elevated blood lipase and amylase may or may not be found but if found is of diagnostic value.

*Acute cholecystitis* may be confused with pancreatitis perforated ulcer or acute appendicitis. A previous history of gallbladder colic and the findings of gallstones in a scout film are of help in diagnosis but all three conditions may be associated.

*Acute pelvic conditions* not only inflammatory but also mechanical such as the twisted pedicle of an ovarian cyst or an ectopic pregnancy often cause symptoms indistinguishable from those of the conditions just described and also may be associated with them.

## TREATMENT

In general it is probably safer to operate upon doubtful cases than to wait. In cases of *perforation* though walling off by adhesions to neighboring organs or covering with omentum may at times obviate immediate operation it is too risky to wait for this since fatal peritonitis might occur.

In *intestinal obstruction* suction through a Miller Abbott tube may at times permit spontaneous reduction of a kink, volvulus or internal hernia. On the other hand relief of distention and pain may lull the patient and doctor into a false sense of security while the blood supply is cut off and the gut becomes gangrenous with the development of peritonitis. In any event the suction should be accompanied by frequent x-ray and laboratory examinations and careful physical check ups at frequent intervals. It should not be continued too long—a few days at most.

Before the decision to operate has been definitely made it is imperative not to do anything that might mask the symptoms. It is best to withhold everything by mouth to give no medication especially cathartics. An enema might prove fatal when perforation of an appendix is impending. If an enema is decided upon to determine whether an obstruction is present it should be small and should be given only in a hospital and by a doctor or a competent nurse. An oil retention enema 5 or 6 ounces of warm oil to be retained can do no great harm in such a case and may soften an impaction and possibly promote reduction of a mild intussusception. Moderate warmth to the abdomen avoiding a burn is safer than an ice bag and satisfies the patient and family. Meanwhile parenteral fluids and electrolytes and possibly transfusion will be of help and will improve the patient's condition should operation be decided upon. Inhalations of oxygen may reduce distention. If time permits some of the laboratory studies may help in elucidating the diagnosis.

After operation has been decided upon adequate preparation may improve the patient's chances of recovery. Now sedatives may be used parenteral injections given as required and antibiotics given to minimize infection at operation. The other procedures recommended for preoperative care should be carried out (p. 232).

The type of operation depends of course upon the nature of the lesion. It is best in an acutely ill patient not to attempt too much at operation lest the patient succumb. Restriction to simple drainage of an abscess, closure of a perforation, enterostomy, cecostomy or colostomy may be lifesaving. When there is any question of the viability of the intestinal wall however it may be better to resect.

Postoperative care should be carefully carried out as recommended on page 54.

### Prognosis

The acute condition within the abdomen is one of our greatest problems. Its care requires careful observation and intimate team work in diagnosis and treatment. It is only by such a program that the high mortality rate can be reduced.

### SUMMARY

In summary, although many of the cardinal symptoms and signs of the acute abdominal condition may be absent in any particular patient, one outstanding characteristic is almost invariably present. This is the feeling of the patient that there is something seriously wrong with him. Even without pain or vomiting, even though the patient is a stoic and tries to bluff himself into thinking he is only imagining himself to be ill, the underlying disease soon makes him realize he is a sick person. He is not satisfied when told by a doctor over the telephone that it is only an "upset stomach" and that he must take milk of magnesia. He or some one near him insists that he requires a careful examination and wants to see the doctor at once, even though it is in the middle of the night.

## Acute Gastroenterocolitis (Food Poisoning)

In a general sense, food poisoning would include all diseases acquired through ingestion with food of a great variety of agents. In this category would be included (1) various organic and inorganic poisons or other injurious substances, (2) bacteria causing also acute or chronic infectious diseases such as pneumonia, typhoid, tuberculosis, cholera, dysentery and others, (3) parasites and their ova causing local or general infestation, (4) foods or drugs to which the patient is allergic.

The term "food poisoning" today is usually applied to poisoning or infection by food which produces acute gastrointestinal symptoms due to acute gastritis, gastroenteritis or gastroenterocolitis. When such acute gastrointestinal symptoms are suspected of being caused by food, the term "food poisoning" should be used only temporarily until investigation has shown the specific etiologic factor. The disease can then be treated scientifically. As many cases are self-limiting and of short duration, the diagnosis is probably made in less than one half of cases. Nonrecognition of the disease may result in chronic changes in the patient's gastrointestinal tract and produce a carrier state, thus ex-

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disturbances (diplopia photophobia mistiness of vision nystagmus sluggish pupils and paralyses) difficulty in chewing and swallowing dryness dyspnea somnolence apathy and general restlessness There is usually no fever Death often occurs within forty eight hours

### TREATMENT

The only specific treatment for botulism is the use of antitoxin for the most common strains It is difficult to obtain and may be of limited value The usual lavages and catharsis must be combined with symptomatic treatment including oral or parenteral stimulation antihistaminic and steroid therapy the use of a respirator preceded by aspiration cardiac stimulants and sedatives as required

### PROPHYLAXIS

Prophylaxis is most important Careful fractional sterilization in the processing of canned and bottled foods and thorough boiling of such foods before eating would prevent botulism effectively The prompt reporting of suspected cases would prevent others from partaking of foods from the same source

### Micrococcus (Staphylococcus) Enterotoxins

Micrococcus (staphylococcus) enterotoxins have been generally recognized only in recent years as a cause of a large percentage of food poisonings They occur at any time of the year a little more frequently in winter Large outbreaks of poisoning usually occur among persons who have eaten in the same restaurant at banquets or suppers or at picnics Many people in a neighborhood may be poisoned by food from one bakery ice cream parlor or delicatessen store The common *Micrococcus pyogenes* usually the *Staphylococcus albus* or *aureus* is most frequently responsible An aerobe it grows readily in any food especially in sauces pies cake fillings salad dressings and cold soups in various milk products in ham and other cold cuts and in jellied chicken The foodstuff gets inoculated by sneezing or nose drippings from a food handler with a cold or by his hands if he has blown or touched his nose and not washed them afterward by contact with an infected wound or its dressing by an infected udder of a cow by organisms adherent to dirty containers used for serving or by any other breaches of elementary sanitary care

After inoculation incubation for several hours is necessary for production of the enterotoxin Although some warmth is necessary for the growth of the organisms they will multiply even at room temperature The food loaded with toxins is not altered in taste smell or appearance Inoculated food standing on a counter in a restaurant in a show window or show case in a store in a lunch basket at a picnic or on a sideboard or window sill in a home becomes a sinister weapon Even if the food

is placed in a refrigerator or cooked in a large container bacterial proliferation in the central portion may go on for hours. If it will destroy the toxin but every portion of the food must be heated to the boiling point for more than fifteen minutes or to 150 °F for thirty minutes in order to make sure. If this is not done the intact toxin at the center of the mass of food may affect the persons who eat this portion whereas the external portions cause no trouble. This accounts for the frequent observation that not all persons eating a certain batch of food are stricken.

### SYMPTOMS

Symptoms occur abruptly within minutes or at most a few hours after ingestion. Vomiting, cramps, profuse watery diarrhea and prostration may occur in a large group even while still at a party. With large numbers trying to get to toilets and standing in line incontinence may occur. Dehydration progresses rapidly. In some individual cases with no other persons near who are similarly affected an impression of "acute abdomen" may be created. On the other hand I have seen patients who really had an acute appendicitis go on to perforation with a false diagnosis of food poisoning. With the poisoning there is no fever or leukocytosis. In any case vomitus, stool and food must be sent to a suitable laboratory for diagnosis. An epidemiological study may prevent a spread of the poisoning. In most cases no further toxins are elaborated in the intestines; the symptoms last only a day or two or at most three or four days.

### TREATMENT

Upon making sure that the case is not one of appendicitis or other acute abdominal calamity, lavage or autolavage, catharsis usually with castor oil, a smooth diet of the ulcer type and soothing medication such as kaolin, pectin or bismuth may be prescribed. With no medication at all however the symptoms will subside in a few days at most. No antibiotics should be used.

Prophylaxis proper care in the handling and sterilizing of food is most important.

It should be noted that this poisoning from the enterotoxins of *Staphylococci* is quite different from the serious staphylococcal infection which frequently follows the use of antibiotics. This is described under Acute Pseudomembranous Colitis (see p. 363).

### Bacterial Food Poisoning

Streptococci and virulent colon bacilli have been implicated in rare cases of food poisoning. Staphylococcal infection and infection with *Shigella* organisms, the latter responsible for bacillary dysentery, are

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discussed elsewhere. The most frequent infection accounting for nearly one third of all food poisoning is due to *Salmonella* organisms of which there are over 300 strains. In general they can be grouped into two types those causing gastroenterocolitis only and those rare ones causing septicemia. In the latter type there may be a more or less severe gastrointestinal tract infection with the organisms absorbed into the circulation producing localized infection in the meninges, lungs, kidneys or elsewhere, a subacute bacterial endocarditis or an ulcerative colitis. Some of the *Salmonellae*, particularly paratyphoid organisms may produce symptoms resembling typhoid fever but usually of shorter duration. Severe infections however may last longer and even result fatally. *Salmonella typhi*, formerly called the typhoid bacillus is of course the cause of typhoid fever. Although as a rule the organisms disappear from the stools in a few days some patients may become carriers for long periods. Rarely *Salmonella* may be the cause of a chronic ulcerative colitis.

### Mode of Infection

The organisms are most frequently food borne. Food gets inoculated through carelessness on the part of food handlers. The manufacturer, dealer or purveyor of food products or the cook or waitress in home or restaurant may be the guilty party. Aside from human carriers, household pets, barnyard animals and fowl may act as reservoirs and the addition of their excreta to food allows the bacteria to propagate. They will grow readily in many foods especially in rich culture media such as custards, ice cream, jellied meats or vegetables, cold cuts, salad dressings or any soft cooked foods allowed to stand about a kitchen or dining room for a time. The incubation period is not long but six to twelve hours is about the minimum time required to produce an infected product. Direct infection of the gastrointestinal tract may also occur as a result of ingestion of milk from an infected cow, eggs from an infected fowl or feces carelessly carried to the mouth from an infected human being or pet. Day old chicks have been the cause of epidemics at Easter time. As a rule where food has become infected epidemics are more apt to occur whereas direct infection produces sporadic cases.

### SYMPTOMS

The gastrointestinal infection causes the usual vomiting, cramps and diarrhea with or without febrile reaction or leukocytosis. The duration is usually from one to five days. The more virulent infections may go on for weeks, the septicemic type may result fatally.

### DIAGNOSIS

In the presence of an epidemic the occurrence of symptoms leads to a presumptive diagnosis. An acute abdomen must be ruled out (see p

59) Stool cultures are usually positive for only a few days except in carriers or chronic cases such as ulcerative colitis. Agglutination and skin tests may be of help although reactions from previous infections may be confusing. The finding of a specific bacteriophage may be of great assistance. Since *Salmonella* infections may at times be serious and the symptoms may simulate those of an enterocolitis due to organic or inorganic poisons, to enterotoxins and viruses or to other infections notably Weil's disease, it is important to report such cases and to have vomitus, stool and foods with their containers sent to suitable laboratories for complete study.

### TREATMENT

Most cases are self limiting and require no more than a preliminary lavage, autolavage, catharsis with castor oil and a bland diet. The use of antibiotics is not usually necessary except in severe septic cases; in the ordinary case it is safer not to give antibiotics especially since their use may lead to development of the serious and often fatal staphylococcal pseudomembranous enterocolitis. The usual kaolin, pectin and other soothing or antispasmodic drugs suggested for diarrheas may be prescribed.

### Virus Infection

Only in the past few years has it been demonstrated that viruses are responsible for many instances of food poisoning "of unknown origin." Fully one third of all cases are due to these microorganisms. During epidemics in institutions Irving Gordon and other virologists made careful studies of the viruses to determine their mode of transfer from patients to healthy volunteers. It was shown that transfer occurred only through ingestion of fecal suspensions. Attempts to infect volunteers by means of ingestion or inhalation of throat washings were unsuccessful. This means that a patient with "virus diarrhea" or "intestinal flu" has invariably swallowed infected feces. This comes principally through contamination of foods or drinks by fecal material on the hands of food handlers who have not washed their hands thoroughly after each of their frequent bowel movements. The incubation period varies from one and one half to five days, averaging two and one half days.

### SYMPTOMS

It has been indicated that these viruses cause symptoms which can be divided clinically into three groups as follows: (1) afebrile infectious nonbacterial gastroenteritis with vomiting, diarrhea and no fever; (2) epidemic nausea and vomiting with upper gastrointestinal symptoms, no diarrhea and little or no fever; (3) febrile infectious nonbacterial gastroenteritis with fever and gastrointestinal symptoms, no diarrhea but with



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prominent constitutional symptoms. The difference in symptoms may be due to infection with different kinds of viruses or to differences in reaction by various patients to the same virus.

The onset of symptoms is often abrupt with anorexia, nausea, vomiting, cramps, watery diarrhea, and mild constitutional symptoms such as head aches, dizziness, and little or no fever. Dehydration may occur rapidly. As a rule, the symptoms clear up spontaneously in one to three or four days. As in other acute diarrheas, patients with previous serious illnesses may succumb to the insult of the symptoms of this virus infection.

### DIAGNOSIS

It is so far extremely difficult to make a definite diagnosis of a virus infection. By the time a laboratory equipped for virus work could identify the virus, the patient would have recovered from his attack. The onset, symptoms, and even incubation period resemble those of the other conditions due to food poisoning. It is therefore necessary to have vomitus, stools, and food examined as for the other types in order to rule out specific bacteria or poisons. A detailed history of the food eaten previous to an attack and the way it was prepared and handled may give a clue to the etiology.

### TREATMENT

The therapy recommended for diarrheas in general is about all that is required. A bland diet should be pushed, fluids given to overcome dehydration, and mild gastrointestinal sedatives and antispasmodics may be prescribed, although the patient really does as well without them. Antibiotics have no effect on viruses and may make serious trouble. Prophylaxis is important as in the other types of food poisoning.

### PROGNOSIS

Although immunity to a virus has been demonstrated, it is specific for only one strain. A patient therefore may have recurrences due to other strains. The immunity may last for only six months to a year.

### Summary

From consideration of the different types of food poisoning, it is evident that prophylaxis is most important. Carelessness in the processing and handling of food is to blame for all cases. When we realize that an attack of acute food poisoning may be the forerunner of a chronic infection, often of serious proportions, or may predispose the intestinal wall to allergic reactions, the importance of these preventive measures becomes evident. Each patient with food poisoning should be carefully instructed about its etiology so that he can become a worker for more stringent enforcement of sanitary laws.

# Gastrointestinal Allergy

## Introduction

Gastrointestinal manifestations of food allergy are undoubtedly as common as those in the skin or in the mucous membranes of the respiratory tract. The proof of the occurrence of such an allergic reaction in the gastrointestinal tract is so easy: the disappearance of symptoms and signs after removal of the offending food or other allergen and the recurrence of the manifestations after reintroduction of the allergen are so dramatic that it is really remarkable how long it has taken to secure recognition of the phenomenon.

Food allergy is not a newly discovered condition. History records many instances of food allergy recognized even by the ancients. Lucretius in the first century B.C. spoke of "one man's meat is another man's poison." Galen in the second century of the Christian era described goats milk allergy. References to allergy can be found in the Bible and the Talmud. In modern times Hinech eighty years ago, Lauder Brunton over sixty years ago and Max Einhorn over fifty years ago wrote about gastrointestinal allergy.

In the past thirty years a vast amount of work has been done in the study of allergy in general and in regard to its gastrointestinal manifestations. At first the reports came largely from clinicians who established the criterion for a diagnosis of allergy, namely, the disappearance of symptoms and signs of a lesion upon withdrawal of a certain factor called an allergen, such as an ingested, inhaled or applied allergen, and the recurrence of these symptoms and signs upon resumption of the allergen. Later reports of animal and laboratory experimentation have appeared in the literature. Though they have not yet established all the exact mechanisms and causes of allergic reactions, they have indicated that these reactions in the body can be divided into two general classifications which can be used for objective evidence of allergy. Two types have been described.

## Allergic Reactions

The *plasma antibody reactions* occur early at the time of union of antigen and antibody in the blood in which histamine apparently plays a part. Antihistamines may reduce the severity of this reaction. A later result of this reaction, the Arthus phenomenon, produces necrosis and hemorrhage associated with edema and cellular infiltration and comes as a result of obstructive intravascular masses formed by agglutination

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prominent constitutional symptoms. The difference in symptoms may be due to infection with different kinds of viruses or to differences in reaction by various patients to the same virus.

The onset of symptoms is often abrupt with anorexia, nausea, vomiting, cramps, watery diarrhea, and mild constitutional symptoms such as headache, dizziness, and little or no fever. Dehydration may occur rapidly. As a rule the symptoms clear up spontaneously in one to three or four days. As in other acute diarrheas, patients with previous serious illnesses may succumb to the insult of the symptoms of this virus infection.

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### TREATMENT

The therapy recommended for diarrheas in general is about all that is required. A bland diet should be pushed, fluids given to overcome dehydration, and mild gastrointestinal sedatives and antispasmodics may be prescribed, although the patient really does as well without them. Antibiotics have no effect on viruses and may make serious trouble. Prophylaxis is important as in the other types of food poisoning.

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From consideration of the different types of food poisoning, it is evident that prophylaxis is most important. Carelessness in the processing and handling of food is to blame for all cases. When we realize that an attack of acute food poisoning may be the forerunner of a chronic infection, often of serious proportions, or may predispose the intestinal wall to allergic reactions, the importance of these preventive measures becomes evident. Each patient with food poisoning should be carefully instructed about its etiology so that he can become a worker for more stringent enforcement of sanitary laws.

manifestations of allergy. Only 12 per cent of the patients with allergy had allergic manifestations however as the sole cause of symptoms the others showing it as a complication or a contributing cause of symptoms. With increasing interest in allergy and with more patients with known allergy consulting me these figures have become much higher. The importance of recognition of allergy can be judged by the fact that 30 per cent of my allergy patients had been subjected to multiple operations many probably unnecessary. A series of forty nine patients had had sixty eight operations. The patients varied in age from infancy to old age and sex incidence was about equal.

#### ALLERGIC MANIFESTATIONS

Though it has long been postulated that in addition to actual physical manifestations in the body there might be purely neuromuscular manifestations without physical changes I do not believe that this is often the case. The so called neuromuscular symptoms such as spasms and motility changes with their attendant symptoms occurring in both the respiratory and gastrointestinal tracts are usually secondary to the physical changes just as muscular spasm may be a prominent cause of symptoms in urticaria or eczema. No tissue in the body is exempt from allergic reactions.

#### PATHOLOGY

Pathological changes in the gastrointestinal tract can be conveniently and graphically compared with those occurring in the skin the difference being due only to the fact that the mucosa instead of the skin is the shock organ. Walzer and his associates first described the gross and microscopic changes seen in the mucous membranes of human beings and of rhesus monkeys during allergic reactions produced by passively sensitizing the mucosa and then feeding the allergen to the subject. The first reaction often occurring in a matter of minutes consists in hyperemia, edema and infiltration usually with eosinophils this stage resembling urticaria. There follows a surface moisture then a pinpoint rash with exudation as in herpes and then with coalescence of the small papules and more infiltration beneath them there occurs a superficial slough with bleeding resembling eczema. These reactions may as in the skin stop at any point in the sequence and clear up promptly leaving no visible trace of their presence. If the reaction is continued and prolonged as a result of frequent or constant application of the allergen the pathologic changes go on to increasing interstitial granulomatous infiltration polypoid changes scarring deformities interference with circulation deep sloughs with ulceration and occasional perforation and fistulation. Frequently these lesions are complicated by secondary infection such as is seen in regional enteritis and ulcerative colitis.

In 1947 in a review of findings in 120 cases of cicatrizing enteritis

and precipitation of antigen by an excess of antibody. Corticosteroids by abolishing or reducing plasma antibody formations may reduce the formation of these masses.

The *lymphocytic antibody reaction* is a much later phenomenon. An example is a tuberculin type of reaction which develops slowly. It is noted in many bacterial and virus infections. It usually occurs eighteen to thirty six hours after entrance of the antigen but may not occur until five to seven days later as in periarteritis nodosa and other collagen diseases. The exact mechanism is not yet established but corticosteroids may prevent or abolish this reaction. Collagen diseases which have been attributed to allergy are discussed in the next chapter.

*Plasma and lymphocytic antibody reactions* may be combined so that symptoms may occur both immediately and later causing confusion in diagnosis and treatment.

### ETIOLOGY

Though the exact mechanism is not known certain facts about allergy have been accumulated. The *hereditary factor* is amply demonstrated in most cases by a careful history and it has been postulated that there is a gene whose transmission is the cause of allergic sensitivity. An *endocrine factor* undoubtedly contributes to the problem as evidenced by the fact that allergic symptoms are often initiated or influenced by profound changes in the endocrines as at puberty, the menopause and during pregnancy and by the effect upon the symptoms of pituitary, parathyroid extracts, epinephrine, cortisone and ACTH. *Food, vitamin and mineral deficiencies and metabolic disturbances* are predisposing factors. It has been suggested that allergy to the patient's own hormones may occur as in menstrual migraine. *Local infections* may play a part in reducing general resistance or may be the site for the absorption of an allergen obtained either from the bacteria themselves or from substances produced at the site of the infection. The allergen-antibody reactions may take place upon direct contact as in the case of ingestants, inhalants or application. More frequently they occur at a distance, the allergen being absorbed and carried in the blood stream to any part of the gastrointestinal tract and its appendages. Absorption of specific unchanged proteins has been shown to occur in a normal stomach, a distant reaction occurring as early as four or five minutes after ingestion of the allergen.

### INCIDENCE

The frequency with which gastrointestinal symptoms can be proved to be due in part or altogether to allergic manifestations is demonstrated by the findings in a survey of 3412 private patients seen in my practice some years ago of whom 522 or 15 per cent gave a history of allergy in general and 376 or 11 per cent had definite gastrointestinal

edematous bowel may be found subsiding during manipulation. Such operations may be avoided if the patient (especially when showing an eosinophilia) is given an intramuscular injection of epinephrine. This may at times cause sudden dramatic subsidence of symptoms.

Chronic generalized allergic reactions are rare but general gastrointestinal symptoms may be a part of the more usual localized chronic manifestations. Prolonged febrile reactions although occasionally due to secondary infections may occur as a result of severe allergic reactions alone with peaks of temperature spiking to 103 or 104° F.

### Local Reactions

Symptoms of allergic reactions occurring locally in different parts of the alimentary canal will vary according to their location. In the *mouth* aphthous ulcers (canker sores) are the most frequent manifestations but gingivitis glossitis stomatitis and cheilosis may often be due to allergy. Other causes must of course be ruled out. In the *esophagus* there may be localized or general manifestations either acute or chronic. Such an esophagitis occasionally with marked indurative and fibrotic changes may be mistaken for cancer. Allergic ulcers or erosions may occur and achalasia (cardiospasm) may at times be traced to an allergy. In each case the typical symptoms of esophageal disease are present and careful study must be made to rule out all other causes. In the *stomach* and *duodenum* ingestion of an allergen may produce transient edema redness and puckering of the mucosa and even erosions. If taken frequently an allergen may produce a real gastritis. Rarely an acute reaction in the pyloric region may cause complete but transient stenosis. These findings can be demonstrated by x ray study and by gastroscopy. Peptic ulcer which in many respects resembles an allergic reaction has not been definitely proved to be due to food allergy but its relation to focal infections suggests the possibility of a reaction to some product probably a histamine like substance absorbed from such an area of infection. Allergy to some food such as milk included in an ulcer diet may hinder normal healing. In the *small* and *large intestines* intermittent mild allergic reactions may cause feelings of peristaltic unrest retrostaltic symptoms or diarrhea often with excessive discharge of mucus. The x ray patterns described as due to subvittaminosis celiac disease or irritable or unstable colon are most frequently due to allergy as may be proved by adding the allergen to the barium mixture. Milk cereal chocolate or other flavoring in the mixture may induce the findings which will not be seen when the allergen is avoided. In the *anorectal* region the reactions may resemble those in the colon or there may be evanescent purpuric areas causing intermittent hemorrhages. The most frequent allergic manifestations are cryptitis anal fissure and pruritus.

(regional enteritis) Shields Warren and Sommers described acute subacute and chronic phases mentioning that the etiology is unknown. Their description of the early phases coincides almost exactly with the findings of Walzer in allergic reactions. Bergmann in 1932 described similar pathological changes which he named "Asthma bronchiale des Dickdarms." Single large or multiple smaller purpuric areas are also usually due to allergy and may be the cause of hemorrhages. They are generally seen in the rectum and sigmoid. Henoch's purpura is usually due to allergy.

In my experience *malignant changes* are rare although they have been demonstrated in the areas of fibrous, granulomatous and polypoid changes. In the biliary tract and appendix the allergic reaction may consist only in edema and round cell (eosinophile) infiltration. In the pancreas an acute pancreatitis may be produced. The collagen diseases are generally supposed to be allergic in origin. Sprue may also have an allergic background.

### SYMPTOMATOLOGY

As allergic manifestations in the gastrointestinal tract often closely resemble organic lesions not only in their symptomatology but even in their physical and laboratory findings it is important to emphasize at the start that a complete study of the patient is absolutely necessary to rule out such organic lesions.

The *history* is most important. A family history of allergy not necessarily of the gastrointestinal tract is of help because allergy is definitely a hereditary condition. One or both parents as well as grandparents or siblings will frequently be found to have had asthma, hay fever, migraine, canker sores, skin or other manifestations in either the past or the present. On the other hand, an acute infection in the gastrointestinal tract or elsewhere may precede the onset of allergic manifestations.

The symptoms vary with the location and severity of the reaction and its duration. If it is *general* and *acute* affecting the entire gastrointestinal tract the symptoms are those of an acute gastroenteritis or food poisoning with fever, malaise and grippy feelings in addition to vomiting and diarrhea. The symptoms are frequently associated with other allergic manifestations such as urticaria, pruritus, migraine or vasomotor rhinitis. Such reactions are often seen in patients allergic to shellfish and are easily confused with actual infections such as *Salmonella* or *Staphylococcus* gastroenteritis which however usually last longer.

*Localized severe acute* reactions with definite edema, induration and spasm may produce the symptoms of an acute abdomen resembling an intestinal obstruction or a perforation (see p. 59). Such patients are often operated upon but either no lesion is discovered or a boggy

## LABORATORY PROCEDURES

Laboratory procedures may be of both direct and indirect value

## Blood Studies

Blood studies may show an eosinophilia which when parasitic infestation can be ruled out is of considerable value as indicating an allergy although not invariably present. The leukopenic index of which much was written some years ago is not reliable. Other blood findings will vary with the extent, location and complications of the allergic reaction. Blood chemistry examination may be useful in evaluating the need for treatment and for food, water and electrolytes. The degree of anemia may be important.

## Gastric Analysis

Fractional gastric analysis may disclose an achylia which was formerly thought to be essential for the absorption of unchanged proteins which cause the allergic reaction. Normal acid secretion is much more frequently present however and its presence has been used experimentally to show that unchanged protein can be absorbed even with normal secretion. The incidental findings such as mucus, blood and bile are of value in diagnosis of allergic manifestations in the stomach.

## Stool Examinations

Stool examinations are usually important especially when intestinal manifestations of allergy are suspected. The repeated studies are used to help in ruling out parasitic infestation, cancer, purulent and other bacterial and mycotic infections. The finding of eosinophils in the bloody mucoid discharge from an ulcerative colitis has been pointed out as an indication of an allergic reaction. Excessive mucus, an indication of irritation, is often due to allergy.

## X RAY EXAMINATIONS

X ray studies may be of great significance. Aside from disclosing the evidences of the organic changes produced by allergy in any part of the gastrointestinal tract, some x ray findings fairly definitely indicate an allergic reaction. The disappearance of evidences of the more severe lesions such as regional enteritis and ulcerative colitis after withdrawal of allergens and the repeated reappearance of these lesions upon resumption of allergens are good evidences of allergy. *Fluoroscopy* of the upper gastrointestinal tract after ingestion of a barium meal containing a substance to which a patient is allergic such as milk or a flavoring or some suspected food will usually disclose a puckering of the mucosa of the esophagus, stomach or duodenum, often with spasm or with changes in motility. Allergy as a cause may be determined by using a mixture



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and any of which may be accompanied by infection and abscess followed by fistula. The *gallbladder appendix* and *pancreas* are occasional sites for localized allergic reactions which may subside spontaneously or may go on to complications.

Repeated or persistent allergic reactions due to frequent or constant exposure to the foods, drugs, chemicals, inhalants or other allergens to which the intestine is sensitized will induce all the stages of the reactions observed by Walzer, finally producing a cicatrizing enteritis or an ulcerative colitis, the two sometimes occurring together and either one showing localized (skip areas) or general involvement. Failure to recognize the cause of these conditions will eventually result in complications such as mentioned under Pathology, as well as food and vitamin deficiencies and fluid and chemical imbalances due to failure of digestion and absorption.

### PHYSICAL EXAMINATION

Physical examination is mainly of importance to discover organic disease in order to rule this out as the cause of the symptoms. Even in organic disease, however, an allergic reaction may be coexistent. Evidences of cardiovascular, respiratory, renal, pelvic, endocrine and hematological diseases must be searched for. Focal infections, mycotic diseases or other manifestations in the mouth, nose, throat, rectum, pelvis, skin and elsewhere must be located so that they can be eradicated or treated.

Careful *abdominal examination* may disclose definite organic lesions or the findings may be equivocal, varying from those of an "acute abdomen" to no evidences of disease.

*Rectal examination* by the finger is important to rule out cancer. Mere inspection will show the pale circumferential induration of pruritus and fissures, fistulas or abscesses which may have originated in an allergic process.

### INSTRUMENTAL EXAMINATION

Instrumental examination may include the examination of the *esophagus* by bougies and esophagoscopy, disclosing the narrowing and the appearance of esophagitis, ruling out cancer by biopsy. The *stomach* may be inspected by gastroscope, showing the gastritis or even ulcer due to allergy, and helping to rule out cancer. The *rectum* and *sigmoid* should be inspected through an anoscope and a proctoscope, revealing in a case of allergy the redness, edema, surface exudation, pinpoint nodules, eczematoid appearance, sloughing and extensive ulceration of the various stages culminating in ulcerative colitis. Typical purpuric areas can also be seen and usually account for hemorrhages. Disappearance of lesions when allergens are withdrawn and reappearance upon their resumption prove allergy as the cause.

in the atmosphere. Allergic symptoms are often also relieved during periods of exposure to sunshine.

3 If a patient with a proved organic disease has symptoms not at all explainable on the basis of the condition found or not responding normally to dietetic or other treatments. For instance, in a patient with peptic ulcer and milk allergy the usual milk diet will not relieve but actually aggravate the symptoms and a patient with an allergy to a drug ordinarily used to relieve symptoms may find that they are aggravated by the drug or that additional new symptoms are caused by it.

4 If the patient gives a history of knowing definitely that certain foods will invariably cause certain gastrointestinal symptoms such as coffee or milk acting as cathartics, chocolate producing heartburn, tomatoes, cinder sores, strawberries and pruritus and other similar sequences. Such a history should never be ignored. It is usually easy to distinguish between a patient's mere aversion to certain foods and real symptoms resulting from them.

### Grouping of Allergens

*Ingestants.* Foods are the most common allergens. Those ordinarily taken *every day* will produce the most violent continuous reactions. They include milk, egg, wheat, potato, orange, tomato, coffee, tea and perhaps chocolate and condiments. Those taken *intermittently* may cause intermittent mild or severe symptoms. These include fruits, berries, nuts, seasonal vegetables, flavorings, specific meats, fish and unusual foods. Some patients are sensitive to milk only at certain times of the year depending upon food substances eaten by the cow or goat differing when in pasture or feeding indoors. The most frequent offenders vary in different parts of the world. In my experience milk is the most frequent food allergen being the cause of symptoms in 60 per cent of cases. Wheat, orange, tomato, egg, chocolate and potato sensitivity occur in about the order named.

*Drugs and chemicals* are often overlooked as causes of allergic reactions and often the drugs given to correct symptoms actually increase them or are the cause of new symptoms. The frequent occurrence of rectal symptoms and pruritus following the administration of antibiotics has already been mentioned. Drug sensitiveness or allergy to belladonna, the barbiturates, laxatives, various coal tar derivatives, arsenic, mercury, iron, bromine and others has long been recognized. Sensitivity to ingredients of dentifrices, lipstick, cosmetics, nasal sprays and perfume and to insecticides adherent to fruit and vegetables must also be considered. In the use of hormone preparations, vitamin concentrates and serums of various kinds it is to be remembered that even when sensitivity to the hormone or vitamin may not be present there may occur an allergic reaction from sensitivity to the animals or vegetables from which

devoid of the suspected allergen with no ensuing reaction. Irritability of any part of the gastrointestinal tract is suggestive of allergy. In a *barium enema* study mucosal changes similar to and often more definite than those seen in the upper gastrointestinal tract are frequently observed. In cholecystographic studies allergy to the "dye" or its iodine constituent may cause abnormal findings suggestive of gallbladder disease and may cause diarrhea or vomiting. Scout films are of value in ruling out perforation, obstruction or calculi. Chest films may detect pulmonary complications.

### DIAGNOSIS

It cannot be emphasized too much that before making a diagnosis of allergy as the cause of gastrointestinal symptoms it is absolutely necessary that a complete and careful general and gastrointestinal survey be carried out in order to rule out organic disease. Too often I see patients with malignant and other lesions which have been overlooked because of an undue enthusiasm about allergy. I have seen several patients with rectal carcinoma within reach of the examining finger who were treated by elimination diets with the mistaken diagnosis of ulcerative colitis. All methods of diagnosis must be used to make sure that nothing has been overlooked. All other causes for the patient's symptoms having been ruled out, the most important step is to determine the factor responsible. It must also be remembered that allergic manifestations may complicate specific diseases or may have preceded them. In some instances allergy may follow acute disease. Regional enteritis and ulcerative colitis due to specific allergens may follow acute gastroenteritis due to bacteria or viruses or may occur after bacillary or amebic dysentery has apparently been cured. To institute general treatment for allergy without knowing exactly what agents are the cause of the patient's disease is bad practice, since elimination of the *cause* offers the only real hope of cure.

Allergy may be suspected if any of the following four conditions are present:

1. If the symptoms are bizarre, not ordinarily explainable on an organic or infectious basis, as for instance, the occurrence of severe symptoms suggesting an abdominal calamity which subside suddenly, either spontaneously or after an injection or epinephrine, with no symptoms or signs of disease remaining. The same is true also to a great extent when symptoms subside promptly after corticosteroid therapy.

2. If the symptoms occur intermittently with no symptoms between attacks. In some cases a specific allergen may be suspected from occurrence of symptoms including hemorrhages on certain days of the week when certain foods are taken, from the season of the year when certain foods ripen or are habitually eaten, when change in fodder of cows may include the patient's antigen or when certain pollens are prevalent.

pointed out a careful history a cooperative patient who has been told what is being searched for and a mind capable of deductive and inductive reasoning are essential. The average busy allergist does not have time for such a study so that when a clinician reports that he referred a patient to an allergist without result it does not mean ordinarily that the patient has had an adequate gastrointestinal allergy study.

**Skin Tests** Skin tests are the first thought of the clinician and allergist alike although most allergists agree that skin tests whether done by the scratch intradermal patch or passive transfer methods are of little or no value in determining sensitivity to foods. They may be useful however in testing for pollens mycoses contact allergens and drugs. The final proof depends on the disappearance of symptoms upon elimination of the suspected allergen and their invariable recurrence upon its resumption even in very small amounts.

**Diet Studies** Diet studies are most reliable but often difficult. It must be borne in mind that the symptoms may appear within five minutes or not until twelve hours or more after ingestion and may last for hours or days after the food has been taken only once being continued as long as there is absorbable allergen in the body. The diet studies can conveniently be carried on while the patient is undergoing a general study. There are in general three methods of diet study each applicable to different types of symptoms as follows:

**DIARY STUDY** The patient keeps an accurate timetable of all foods drinks condiments and other substances ingested any extraneous factors which might have a bearing on symptoms the time and duration of all symptoms and the time and character of all stools. This method of study is mainly applicable in cases of intermittent attacks of symptoms either mild or severe but with intervals of freedom from symptoms. Occasionally an intelligent patient to whom the principles of allergy have been explained can without keeping a record determine the cause of such attacks by simple observation. Sometimes the elimination of a suspected food such as milk in any form may produce a dramatic disappearance of symptoms making even a diary study unnecessary. I have found it of great assistance in making a diary study to explain to patients in detail the principles of allergy and the methods of determining to which food they are sensitive. The following is a copy of the instruction sheets which I give to each patient at the time.

### Instructions to Patients

The cause of allergy is not known but much is known about its nature. There is no cure but avoidance of foods or other factors such as medicines or inhalants known to cause symptoms will prevent these symptoms. There is a variation in sensitivity to foods at different seasons and patients may lose sensitivity to some foods altogether and become sensitive to new ones at any time.

Allergic symptoms include pain cramps burning nausea vomiting gas diarrhea constipation headache rash itching bleeding various nervous symptoms.

they are derived. Pork sensitivity accounts frequently for reactions to hormones derived from pork. Fish, yeast or wheat sensitivity may produce reactions after ingestion of vitamins obtained from these. Horse or other animal sensitivity may account for serum reactions.

*Inhalants* Material inhaled and absorbed through the mucosa of the respiratory tract or swallowed in small amounts may account for allergic reactions in the gastrointestinal tract. Sensitivity to such common inhalants as tobacco and other smoke, dusts, illuminating gas, automobile exhaust, perfumes, cosmetics, sprays and volatile solvents as in paints must be considered. I see many patients whose gastrointestinal symptoms occur each year at the season when pollens are producing hay fever and these are often helped by desensitization. Some patients exhibit *only* gastrointestinal manifestations at that season, the symptoms varying from mild gastrointestinal "unrest" to typical peptic ulcers or severe ulcerative colitis.

*Bacteria and Parasites* Allergy to bacteria has already been mentioned. The problem of allergy and susceptibility to infection involve similar situations and may be difficult to differentiate. Intestinal parasites may also be the cause of allergic reactions as may mycotic diseases.

*Contacts* Contact allergy, sometimes called atopy, primarily affecting the skin, may conceivably cause distant reactions if the allergen is absorbed. In the gastrointestinal tract, as has been pointed out, contact with the mucosa is one cause of reactions, the other being absorption from the skin or mucosa and subsequent reaction due to the blood carrying the allergen to the affected tissues. In the rectum and sigmoid, direct contact with the substances introduced through the anus, such as suppositories, ointments, enemas or instillations may result in reactions.

*Parenteral Medications* Parenterally introduced drugs, chemicals, hormones, vitamins, proteins, serums, vaccines and other preparations may cause reactions as may substances injected intradermally for allergy testing. Exacerbations of ulcerative colitis due to milk allergy may frequently follow the injection of blood from a donor drinking milk.

*Physical Allergy* Physical allergy, so called, including sensitivity to external heat, cold, climatic and electrical factors, may play a part. Nausea or diarrhea may result from heat, intestinal cramps from cold.

### Determination of Allergic Factors

Determination of the allergic factors is not easy. It has been so discouraging to many clinicians that they have given up in despair and deny the mere suggestion that allergy can cause gastrointestinal symptoms. They therefore descend to the much easier explanation of psychosomatic disease. Practically all my patients have previously been treated on this basis and many have had elaborate psychiatric treatments, psychoanalysis, shock therapy and even institutional care. As already

This test diet is applicable to cases in which symptoms are present every day including such mild daily symptoms as nausea, heartburn or abdominal pains and such severe conditions as regional ileitis and ulcerative colitis. This diet consists of six foods only: gelatin, sugar, two cereals, one or two vegetables and one fruit.

The cereals, vegetables and fruit are chosen after detailed consultation with the patient and only those probably not responsible for symptoms are used. Rice, rice peas and raspberries or grapes are particularly desirable and easily taken. The berries are taken plain or as pure jam or juice. An example of such a diet is as follows:

### Allergy Diet No. 2

Eat only the following foods: Rice, rice, carrots, peas, raspberry and gelatin.

#### *Breakfast*

Dish of rice—boiled or as dry cereal, with water and sugar  
Ry Krisp with raspberry jam  
Dish of raspberries with sugar  
Gelatin drink

#### *Lunch*

Peas  
Boiled rice  
Peas and carrots  
Ry Krisp with raspberry jam  
Raspberries, plain or in jelly  
Gelatin drink

#### *Supper*

Same as lunch or breakfast

#### *Between Meals and at Bedtime*

Gelatin drink  
Ry Krisp with raspberry jam

Gelatin should amount to 50 to 75 gm. a day (six or eight envelopes of Knox Gelatin). This can be made into a jelly or can be mixed with or merely stirred into raspberry juice.

Sugar, either dextrose or saccharose, can be used freely, remembering, however, that dextrose may cause diarrhea.

Obviously, this is not a well balanced maintenance diet, and this fact must be explained to the patient. It cannot be followed for more than a few days, especially in a badly depleted patient. In ulcerative colitis patients I frequently precede it by a couple of transfusions and some parenteral alimentation for a few days. It must be followed closely. The patient must keep an exact record of the time and character of the stools, and the physician and attendants must look for even minor improvements from day to day. If symptoms are not promptly alleviated, changes will have to be made in this diet as indicated by careful study of the reactions in relation to items in the diet. Within three or four days, if the diet contains no food to which the patient is allergic and all other

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started to use this diet many years ago for gastrointestinal allergy and found it a valuable procedure but I have enlarged it by using six foods as a basic diet for the preliminary period. I use two such diets which I have called Allergy Test Diets numbers 1 and 2.

Allergy test diet number 1 is used when the patient's symptoms occur in short attacks such as colics, diarrheas, migraine, vomiting spells and the like which last only a day or a few days and are succeeded by periods of freedom from symptoms. Logically such attacks must be due to foods taken only at intervals and a basic diet should consist only of foods taken daily by the patient. I allow milk, egg, gelatin, wheat, sugar, potato and orange in this diet, substituting some other cereal, vegetable or fruit if the patient, knowing that any of them has been causing symptoms, has been avoiding them. By combining only these six foods at three meals and at between meal and bedtime feedings, a well balanced nutritious diet can be formulated. Such a diet would be as follows:

### Allergy Test Diet No. 1

Eat only the following foods: Milk, egg, wheat, sugar, potato, orange and gelatin.

#### *Breakfast*

Dish of wheat cereal with milk and sugar

1 or 2 soft boiled or poached eggs

Wheat bread and butter

1 glass of milk

Orange

#### *Lunch*

Egg or cheese (cream or cottage)

Baked or mashed potato

Wheat bread and butter

1 glass of milk

Orange custard, wheat (bread) pudding

#### *Supper*

Same as lunch or breakfast

#### *Between Meals and at Bedtime*

1 envelope of gelatin mixed with water, swallowed rapidly

1 glass of milk

Wheat bread and butter or wheat crackers

When, after a few days, this diet has been demonstrated to cause no symptoms, daily additions of one or two foods, beverages, flavoring or condiments should be made. Such additions suspected of causing symptoms must be rechecked to establish their specificity. A sudden violent exacerbation of symptoms or the occurrence of a new train of symptoms upon starting this diet indicates that one or more of the six ingredients of this diet are the cause. The indication then is either to eliminate one food at a time and watch for results or to change to diet number 2.

Allergy test diet number 2 eliminates all foods habitually included in the patient's daily menu and consists of six foods not taken frequently.

### Diet

Just as patients must know which foods they are allergic to and which they must avoid so it is imperative that they be told exactly what and when they are to eat. The diet should list all items to be taken at each meal at between meal feedings and at bedtime. In formulating a diet *a proper balance of ingredients as well as suitable caloric value must be maintained*. In addition to a proper proportion of proteins, carbohydrates and fats, adequate vitamin, mineral and water content must be provided. Gelatin is a valuable adjunct to the between meal feedings, providing protein, calcium and a coagulant effect in case of hemorrhage. Parenteral feedings not containing the allergens discovered may be required for a time. Blood transfusions may be necessary, but it is essential that the blood donor for at least three days before his blood is taken should avoid eating the food to which the patient is sensitized.

### General Care

Coincident with elimination of all allergens responsible for the patient's symptoms, it is important to treat the patient as a whole, as an individual differing from all other individuals. As mentioned before, all areas of focal infection should be promptly eradicated and adequate care of respiratory, circulatory and renal disease should be instituted. Mycotic infections and parasitic infestations must be eliminated. Anemias, food deficiencies and psychologic complications may require special attention also; the patient's family needs encouragement and explanations of the patient's findings and treatment.

Complications in the gastrointestinal tract are often difficult to treat. Coincident or secondary infections and infestations may require chemotherapy or antibiotics, bacteriophages, vaccines or serums.

### Medication

In general, medication is to be avoided as much as possible. I see too many patients in whom medication has been insisted upon, even though the patients themselves at times realize that they are being harmed by it. In an allergic patient there is always the danger that *sensitization to drugs* may exist or be acquired, and I find it safest not to use medication except perhaps occasional temporary sedation or the use of antispasmodics or antidiarrheal remedies. *Antihistaminics* are rarely of any use in gastrointestinal allergy, and many patients may show definite allergic reactions to the drugs themselves. Occasionally, however, in mild cases an antihistamine taken with food to which a patient is known to be allergic may prevent an allergic reaction. In some cases Benzedrine or Dextedrine may provide some relief of symptoms. In many cases some long term benefits may result from calcium, either alone or combined with



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causes of symptoms have been ruled out the patient usually shows some improvement. Pains and cramps become less severe and frequent. Bowel movements are reduced in frequency and looseness and the stools become less explosive and irritating. In general the patient looks less toxic and more alert and says he can feel some general improvement. It is then safe to commence with daily additions of the diet, avoiding for a couple of weeks any foods which have been suspected of causing symptoms. I usually start with a steak or chicken, add other proteins for one or two days and then fruits and vegetables. Within a week or ten days a fairly good maintenance diet can be attained. Thereupon the resumption of an offending food even in minute amount will often produce a dramatic exacerbation. I observed ten drops of milk given in an attempt to desensitize a patient produce a three day attack of profuse bloody diarrhea when his ulcerative colitis had practically cleared up on a milk free diet.

### TREATMENT

After determination of the specific allergen or allergens causing the patient's symptoms the first essential part of the treatment logically consists in eliminating them entirely, if possible. With some inhaleds such as dusts and pollens this may not be practicable and resort to desensitization by hypodermic injection of gradually increasing amounts may prove effective. Most of the other substances causing allergic reactions may be entirely avoided by the patient although with some foods this may be difficult. For instance milk, egg and wheat are present in so many foods in an ordinarily normal diet that it is extremely irksome to avoid them entirely. However I have many patients who live a normal life, are well and happy even though they must avoid one or more of these foods.

### Desensitization

Desensitization can be tried but in my experience is rarely successful beyond allowing small amounts to be taken. Some patients appreciate the allowance of a few teaspoonfuls of milk a day. The procedure need not consist in initial hypodermic injection of extracts but can be simple enough the patient starting with ingestion of small amounts of the food in question and increasing the amounts each day until a reaction occurs and then going back to the quantity which did not cause a reaction and not adding to it until later. The initial daily doses I have found to be practicable are one drop of milk, a drop of a mixture of one raw egg beaten in a glass of water or a small pellet of bread. It must be realized that with any form of desensitization the substance must thereafter be taken daily to prevent recurrence of sensitization. This excludes unusual foods or those not able to be taken daily by the patient from the desensitization program.

*The Stomach and Duodenum* Both ulcer and gastroduodenitis require soothing diets with frequent feedings. Balanced rations with avoidance of foods to which the patient is sensitized are relatively easy to formulate. Almost any foods may be responsible for reactions. Surgery is rarely necessary, being required only for complications such as obstruction or perforation.

*The Small and Large Intestine* Even when diarrhea and bleeding are of alarming proportions, the diet need not necessarily be devoid of so-called roughage as long as the known allergens are not included in it. Allergy test diet number 2, with plenty of green vegetables and fruits, usually results in rapid disappearance of even severe manifestations in ulcerative colitis and regional enteritis. Transfusions are usually necessary in the severe cases and parenteral feedings are of help provided the precaution has been exercised against injecting blood or food concentrate containing the allergens. In case of excessive rectal bleeding, instillation of 150 to 200 cc. of hot 5 to 10 per cent gelatin solution often stops the bleeding through its coagulant and soothing effect. Operation may of course be required when obstruction, cicatricial or fibrotic deformity or perforation has occurred, but should be deferred if at all possible to observe the effect of allergic treatment. Even moderate polypoid changes may clear up entirely, although it may take several years to observe their disappearance. As an excuse for the resection of involved intestine in ulcerative colitis and regional enteritis, it has been pointed out that removal of the shock organ will cure the condition. Shock organs tend to change, however, with removal of one, another may occur.

*The Anus and Rectum* There is a tendency routinely to use local applications for the lesions in this region. Often the condition is made worse by the suppositories, ointments, instillations and enemas because the patient may be sensitized to their ingredients. It is usually best to keep the area clean and dry or, if it is thought the oil would be desirable, to use only plain mineral oil or petroleum jelly. It is interesting to note how quickly pruritus ani, fissures, cryptitis and papillitis will clear up when specific foods causing these conditions are eliminated from the diet. Dilatation or operation may at times be required.

*The Gallbladder* Acute gallbladder colic can be obviated by avoiding the allergen and instituting biliary drainage by frequent feedings with adequate fats and oils, but complications such as calculi require suitable care, including surgery. When repeated allergic reactions are confined to the gallbladder and appendix, it would be conservative to remove them.

### Prognosis

Realizing that allergy is a hereditary constitutional condition of unknown etiology, no person can be said to be cured of this condition.

parathyroid extract Corticosteroid therapy so helpful in other manifestations of allergy has not proved to be of lasting benefit in gastrointestinal allergy, although at times there may be dramatic temporary relief of symptoms even in such a severe condition as ulcerative colitis There is however the danger of causing perforation of the colon or producing a peptic ulcer *Corticosteroids, natural or synthetic are contraindicated in tuberculosis peptic ulcer hypertension diabetes osteoporosis and in psychoses* Also when long continued cortical atrophy may result They should be used for but a short time and water retention and loss of potassium should be watched for *Never therapy is occasionally of temporary value in relieving symptoms*

The acute generalized *allergic reaction* in the entire gastrointestinal tract should be treated like food poisoning It is necessary to be sure that one is not dealing with an acute abdomen since appendicitis cholecystitis and pancreatitis may frequently be accompanied by the same kind of pain vomiting diarrhea fever and prostration and may in fact be caused by an allergic reaction A single dose of epinephrine 0.8 to 1 cc of 1:1000 solution given hypodermically may as mentioned before provide dramatic proof of an allergic etiology a high eosinophile count in the absence of parasitic infestation may help to confirm it The indication to get rid of the offending food is best met by a dose of castor oil followed by a kaolin preparation The use of opiates antispasmodics and bismuth preparations is usually unnecessary and may even result in delaying the expulsion of the noxious substance Early frequent feedings of bland foods avoiding any known or suspected allergens will hasten recovery from the attack

### Treatment of Specific Areas

The treatment of allergic manifestations in specific parts of the gastrointestinal tract must often not only include the withdrawal of specific allergens and the measures just mentioned but also must take into consideration the pathologic physiology of the area involved The treatments of each condition are discussed in the chapters devoted to each region

*The Esophagus* A temporary or more rarely a more prolonged obstruction will be produced by the allergic reaction esophagitis or ulcer This would require not only soothing but also liquid feedings which will easily pass the narrow point In patients not sensitized to them milk eggs cream and dextrose mixtures with added gelatin will provide balanced food intake In patients sensitized to milk and eggs gruels sweetened with dextrose with added gelatin and protein concentrates not made from milk or egg may be used Parenteral feedings may be required Demulcents oils and possibly dilutions with bougies or one of the water or mechanical dilators must be considered and in the rare cases of organic obstruction operation may be necessary

6 Complications may occur in severe cases and may require operative procedures

7 The final criterion of allergy consists in the disappearance of symptoms upon withdrawal of the suspected allergen and their recurrence upon its readministration

8 Treatment consists in permanent elimination of the causative factors providing for adequate nourishment and treating complications as required

More detailed consideration of allergic manifestations in the various parts of the gastrointestinal tract will be found in the chapters discussing diseases of each organ

## Collagen Diseases

Since the term "collagen diseases" was first coined by Klemperer in 1942 much notice has been taken of these diseases and much has been done in the way of studying the various phenomena included in this category. The diseases ordinarily included under the term are rheumatic fever, rheumatoid arthritis, lupus erythematosus, scleroderma, dermatomyositis, polyarteritis nodosa and serum sickness.

### PATHOLOGY

The common pathological findings in this group of diseases which show a complex alteration of connective tissues have been described as (1) swelling of the mucoid ground substance, (2) the development of highly refractile and deeply eosinophilic collagen fibers and (3) varying degrees of fibrinoid degeneration and necrosis and the proliferation of fibroblasts. In the late stages fibrosis may occur. It has been suggested that collagen disease may be one of the precursors of cancer. Associated with the connective tissue injury there is usually an elevation of globulin in the blood. At first alpha globulin and later gamma globulin are increased with a corresponding decrease of albumin and an increase of fibrinogen.

### PATHOGENESIS

It has been pointed out that there is not necessarily a common cause for these changes. They could be due to variations in anatomic physiology and chemical factors to substances in the blood resulting from infection or allergy or to endocrine or nutritional imbalance. Allergy has

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Although much can be done to prevent or alleviate specific manifestations. However *several things may happen to an allergic patient*

1 Allergic reactions may cease entirely in one part of the body, often at times of endocrine stress, as at puberty, the menopause or during pregnancy, and immediately or subsequently appear in some other part. For instance, a patient may cease to have hay fever, and develop migraine, and upon its disappearance may show gastrointestinal manifestations.

2 Successively different parts of the body may be involved in allergic reactions at the same time so that a patient may exhibit at one time such reactions as asthma, migraine, urticaria, erythema nodosum, keratitis, and ulcerative colitis.

3 When specific allergens have been determined as the cause of given manifestations and have been eliminated with complete recovery from the condition, the symptoms may recur later, owing to sensitivity having been acquired to some new food or other allergen.

4 The patient may, without apparent reason, lose his sensitivity to certain foods, and either cease to have his allergic reactions altogether or after a time may again become allergic to the same factors or to new ones, with the same symptoms as before.

5 The allergic patient may lose some of his sensitivities after prolonged exposures to the sun, only to have them recur after these exposures have ceased.

6 A patient who has been well for some time may show recurrences after acute generalized or localized infections or even after severe emotional stress. On the other hand, acute or chronic manifestations may disappear after an acute febrile reaction due to any cause or after some major operative procedure.

It is therefore evident that the allergy patient must not only be instructed to watch carefully for the cause of any allergic reaction that may occur at any time in his life, but also should have an occasional conference on the subject with a clinician familiar with allergic manifestations.

### Summary

- 1 Gastrointestinal manifestations of allergy are common.
- 2 They may be caused by contact or by ingested, inhaled or injected allergens.
- 3 Manifestations are essentially mucosal, but may involve the entire wall and may produce neuromuscular manifestations.
- 4 Symptoms may be localized in one part or involve the whole tract and may vary from mild, intermittent gas pains to severe bloody diarrheas.
- 5 The diagnosis of allergy should be made only after a complete study to rule out other organic causes of symptoms.

to use them in any gastrointestinal conditions because of their tendency to cause hemorrhages or perforations. Vitamin C (ascorbic acid) has been shown to be closely related to adrenal cortical hormones. It has an important effect on the production and maintenance of connective tissue and its use in more than mere maintenance doses is indicated. One gram or more per day in divided doses is beneficial and easily tolerated. The citrus bioflavonoids (vitamin P) are a useful adjunct in doses of 200 mg. a day. An adequate balanced diet (p. 21) with added mixtures of vitamins and minerals is necessary as a supplement to the ascorbic acid therapy.

#### COMMENT

The term "collagen disease" is still rather loosely applied to various conditions which are more or less closely related in that they all manifest dysfunction of the connective tissue. As the nature of these diseases is better understood the term may be discarded and more specific names applied.

## Sprue Syndrome (Malabsorption Syndrome)

Formerly considered a tropical disease of unknown origin, sprue is now known as a complex metabolic disorder, a syndrome including both tropical and nontropical sprue, celiac disease and so called idiopathic steatorrhea. Essentially the syndrome is the result of impaired absorption of fat and fat soluble vitamins and also of some carbohydrates, especially polysaccharides and of proteins. The childhood form, *celiac disease*, and the adult form of *idiopathic steatorrhea* appear to be due to hereditary factors. The cause of the secondary form is not adequately understood although nutritional deficiencies resulting from improper diet, allergy, alcoholism, mental and physical stress and infectious diseases undoubtedly play a part, especially in tropical countries. Sprue may be a feature of any prolonged diarrheal disease such as enteritis, colitis and cancer and is also encountered in Whipple's disease, amyloidosis and tuberculosis.

#### PATHOLOGY

The pathological findings are not characteristic but the mucosa of the intestines may have a velvety appearance due to edema; the villi may be

been most frequently suggested as a cause but has not been proved in all types of the lesions. The one common factor suggested as the cause of many of the manifestations has been held to be a vasculitis often called "allergic vasculitis." The fact that adrenal steroids and corticotropin exert a definite beneficial effect upon practically all collagen diseases and also upon allergic manifestations is a possible indication of their relationship.

### GASTROINTESTINAL MANIFESTATIONS

Though polyarteritis nodosa and scleroderma are the only manifestations commonly included among collagen diseases which have been observed in the gastrointestinal tract mucosal ulcerations showing the pathological changes noted above have been observed in lupus erythematosus and dermatomyositis. A number of allergic manifestations have been suggested as suitable for inclusion among collagen diseases. The microscopic appearance of regional enteritis ulcerative colitis pruritus ani and other allergic phenomena and of peptic ulcer could easily be attributed to allergic vasculitis. The course of the gastrointestinal diseases mentioned their tendency to remissions after apparent cure and the effect on them of steroid therapy are similar to those described as more or less characteristic of other collagen diseases.

### SYMPTOMS

Among the symptoms and signs which have been described as occurring in collagen diseases in general are included such gastrointestinal manifestations as purpura hemorrhages and impaired liver function also observed in allergy. Until the concept of what constitutes collagen diseases is clarified it may be somewhat helpful to think of collagen disease when considering at least some of the gastrointestinal manifestations of allergy such as ulcerative colitis and regional ileitis. Peptic ulcer and pancreatic necrosis also show local fibrinoid collagen damage and may later be included among collagen diseases.

### DIAGNOSIS

Collagen disease is not an entity and is recognized today largely because certain manifestations of disease are recognized as belonging in the collagen group. An elevated blood globulin especially gamma globulin is suggestive although it is of course also a finding in other conditions notably in any condition producing an increase in antibodies in the blood.

### TREATMENT

Whereas the use of adrenal corticosteroids and corticotropin is of definite value in most of the collagen diseases it is risky even dangerous

serum calcium is low. Cultures and search for ova and parasites must not be neglected.

### Urine

There are no characteristic findings in the urine except those associated with a complicating nephrosis.

### Blood Tests

Blood tests cannot be used to establish the diagnosis although there is usually a severe anemia frequently the typical picture of pernicious anemia. Low calcium and phosphorus findings low protein with albumin particularly low and hypoglycemia are usual findings. In children acidosis and dehydration occur in "cholera crises." Glucose and vitamin A tolerance tests either give a low curve or show a flat line. Serum creatinine is low or absent. Serum amylase and lipase are normal.

### Gastric Analysis

Gastric analysis after histamine stimulation usually shows a low normal curve of free acidity. This finding would help to differentiate sprue from pernicious anemia.



Figure 8. Malabsorption syndrome. Barium meal study with fragmentation of barium throughout small intestine (deficiency pattern) and moulage sign (sausage shaped smooth area of small intestine (indicated by M)). (Used through the courtesy of Herbert Friedman, M.D.)



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pigmented and deformed the glands may appear trabeculated and the lamina propria infiltrated

### SYMPTOMS

Diarrhea is usually present with frequent bulky homogeneous greasy foul smelling tan stools. Because of faulty nutrition there occurs progressive weight loss anemia often of the Addisonian type (due probably to lack of absorption of vitamin B<sub>12</sub> and folic acid) and skin and mucosal lesions. Edema ascites and hydrothorax may result from the protein deficiency. The symptoms of multiple vitamin deficiencies may include scurvy skin and mucosal hemorrhages tetany pellagra like symptoms spontaneous fractures from osteomalacia and icterus from hepatic damage. Clubbing of fingers is also seen. In mild cases the symptoms may be hardly noticeable and diarrhea may be mild or absent although stools are usually foul smelling. The cause of anemia and malnutrition may be in doubt until a laboratory study is undertaken.

### EXAMINATIONS

#### Physical Examination

Emaciation pallor oral lesions abnormal pigmentations clubbing of the fingers and often great abdominal distention are the principal findings in typical cases. The pigmentation may resemble that of Addison's disease as may the low blood pressure. Neurological findings aside from tetany include paresthesias absent or weak reflexes and occasional ataxia or pseudotabes. The conditions just mentioned under symptoms should be looked for. There is no one test pathognomonic of sprue.

#### X ray Examination

X ray films may show nothing characteristic. The so called deficiency pattern of the small intestine is also seen in allergy (see Fig 8). Kantor also called attention to the moulage sign which is frequently seen the intestinal appearance being that of a moulage. A complete x ray study chest as well as gastrointestinal should be undertaken to determine if possible an organic cause for the deficiency.

#### Stool Examination

Neutral fats constitute from 25 to 35 per cent of the weight of the stool. Repeated estimations of total fats of vitamin A tolerance or absorption of isotopic neutral fat or fatty acids may be used as a guide to the efficacy of treatment. Undigested meat fibers and starch granules are not as numerous in sprue as in the steatorrhea of pancreatic deficiency. Fatty acids and soaps are often found in excess. Calcium is increased when

*Hormone therapy* may be of benefit. *Gonadal therapy* with extra vitamin D will help in cases with decalcification, osteomalacia or osteoporosis. *Corticosteroids* produce definite but often only temporary improvement and may be of help in early stages and in cases resistant to other therapy. The synthetic *prednisone* 15 to 25 mg a day is the preferred form in that salt retention and edema are avoided. This form of therapy should be reserved for severe, prolonged or intractable cases of sprue.

#### PROGNOSIS

This symptom complex is a serious one. It may usually be prevented by early adequate study of the causes of a patient's general malnutrition. Even severe cases are today fairly successfully treated and do not necessarily cause chronic disabilities. In studies of large groups of patients, however, especially of children who have become symptom free, many have been demonstrated to have definite impairment of function. Such patients can be considered to be in a latent phase of the disease and often show a tendency to recurrence after severe stress or disease of any kind.

## Displacements of the Gastrointestinal Tract

### General Discussion

The whole or a part of the gastrointestinal tract may be displaced or any part may be in an abnormal relation to other parts of the gastrointestinal tract or the surrounding organs. The factors causing the abnormal position of the organs may be congenital or acquired and in general include visceroptosis, adhesions, abnormal pressure and herniations. In appraising the findings in any patient with gastrointestinal symptoms, especially if no organic cause of the symptoms has been found, it may be rewarding to make a careful study to rule out displacements. Although in general these conditions may not cause any symptoms, they may at times be the sole cause. The symptoms may be due to retrostalsis or reflex irritation or may be the result of temporary or complete obstruction. Physical examination may not show any findings indicating their presence or may by itself make the diagnosis. In general, x-ray studies are of great assistance in making the diagnosis. The treatment will vary from only diet and hygiene to surgical operations.

## Examination of Duodenal Contents

Pancreatic deficiency may be ruled out by examination of duodenal contents for pancreatic enzymes (see *Pancreas* p 582)

## Other Studies

*Allergy* studies should be thoroughly carried out Allergy to cereal grains especially wheat may be the cause of the syndrome The improvement noted on a gluten free diet suggests that there may be an allergy to gluten

Mild or latent sprue without noticeable diarrhea may be discovered when tetany osteomalacia and unexplained anemia call for further study Examination of the stools may provide the answer

## TREATMENT

*Prophylaxis is most important* In any chronic wasting disease especially one associated with diarrhea proper attention to diet vitamins and electrolyte balance should prevent the development of sprue A careful cleaning up of any focal infections is also important The treatment of any disease which may be the cause of the syndrome must be thoroughly carried out and this will usually result in a gradual improvement in the secondary type

In either the primary or secondary type the treatment must consist of measures to improve the general nutrition The diet usually recommended has been unbalanced consisting of high protein content 120 to 150 gm a day with little or no fat and small amounts of sugars In children the so called banana diet has been popular In adults proteins largely in the form of lean beef ground chicken egg white and cottage cheese and carbohydrates in the form of well cooked cereals and dextrose have been extensively prescribed with later additions of skim milk bread and other starches Recently a gluten free diet has produced prompt improvement in many cases The most important thing however is to find and avoid all food allergens and to prescribe a diet according to the directions in the chapter on Allergy (p 89) A well balanced diet is essential

*Supplementary vitamin and mineral therapy* is necessary Therapeutic doses of all vitamins with fat soluble vitamins A D and K in water soluble form can be accomplished primarily in severe cases by the use of parenteral injections and later in the form of two or three per day of the therapeutic capsules of any reliable drug house Injections of vitamin B<sub>1</sub> combined with folic acid by mouth help the anemia Iron and other minerals can be prescribed in capsule form but may need to be used parenterally at first Transfusions should be avoided if possible Parenteral fluids glucose and electrolytes are required in severe cases potassium being frequently necessary

in women wearing tight corsets now more in men wearing tight belts because suspenders are no longer fashionable. The whole situation is made worse by a stooping slouching posture in children and young people and by failure to exercise the chest in an effort to widen the costal angle. This widening definitely can be accomplished in children when the narrowing is noted and may require removal of upper respiratory obstruction to deep breathing by enlarged tonsils adenoids and nasal obstructions.

The first five factors are all more or less developmental and are primarily influenced by posture exercise diet and by adequate care during and after childbirth. Secondly wasting diseases may cause loss of fat and muscular tone pregnancy will cause more or less weakening of the abdominal wall and pelvic floor sudden removal of abdominal fluid or of a tumor will cause weakening of all supports and of course injuries especially when accompanied by a sudden jolt may dislocate one or more organs.

#### SYMPTOMS

As has been mentioned there may be no symptoms whatever. The gastrointestinal tract adjusted from birth to its lower position due to skeletal conformation usually inherited can function normally. In the acquired type due to neglect of the factors mentioned some ptosed organs may present symptoms which will be discussed.

In general visceroptosis is merely a part of a general asthenic habitus. With adequate prophylaxis in childhood the adult may never experience any of the symptoms of this habitus. The asthenic child is usually peevish and demanding does not eat well and does not develop normally. If not handled properly the resulting symptoms in adult life often classed as neurasthenia include general weakness sleepiness and insomnia headaches or pressure feelings in the head anorexia insufficient and improper food intake resulting in constipation backaches and depressed feelings. At times some of these symptoms may be relieved by upward pressure on the abdomen. These symptoms undoubtedly are due to a combination of food and vitamin deficiencies endocrine imbalance neuroses or actual psychoses and often of real organic diseases of the gastrointestinal tract and not attributable primarily to the visceroptosis.

Symptoms have been attributed to ptosis of different organs and may at times include the following:

*Gastroptosis* together with associated ptosis of the duodenum and jejunum may cause epigastric discomfort nausea eructations fullness and a feeling of downward dragging especially after eating relieved by upward pressure against the abdomen and by lying down especially with the hips elevated. With marked gastric atony vomiting may occur.

Each of the conditions associated with abnormal position of the parts of the gastrointestinal tract will be considered separately

### Visceroptosis

Visceroptosis or splanchnoptosis displacement downward of one or more of the abdominal viscera was a frequent diagnosis in the first quarter of this century. Ptosis intestinal atony and intestinal toxemia were a triad used to explain the symptoms previously described as "nervous dyspepsia and indigestion" and in fact were given as the reason for neuroses in general and even psychoses. The gastroduaphane and the early experiments with x rays were used mainly to demonstrate ptosis and later most x ray diagnoses included mention of ptosis. Many treatments including long periods of rest in bed in the Trendelenburg position various kinds of abdominal supports and surgical operations such as fixation of organs in supposed normal position shortening of ligaments and even excision of ptosed organs were carried out. One great difficulty in making the diagnosis was that no one had fixed a definite level to which an organ would have to drop to be called ptotic. This stimulated a tremendous amount of research which finally disclosed definitely that there seemed to be no degree of so called ptosis which could not be found in persons who were without symptoms and whose gastrointestinal function was normal. Gradually the diagnosis has faded out of the picture so that today it is unusual to find any mention of visceroptosis in the final diagnosis of a radiologist even though a low position of some organ is described among the findings. The organs found most frequently in a low position are the stomach the colon and the right kidney.

### SUPPORT OF VISCERA

Normally the viscera are held in place by a number of factors (1) ligaments mesenteries and mesocolon hold them to the posterior peritoneum (2) pressure of the abdominal wall (3) negative pressure from the chest holding the diaphragm up (4) retroperitoneal mesenteric and omental fat (5) the pelvic organs providing a floor for support (6) the shape of the bony structures forming the abdominal cavities into the shape of a truncated cone with the narrow end down.

The most important factor is the sixth the shape of the skeleton. The reversal of the truncated cone is a part of the asthenic habitus. The upper part of the abdominal cavity is narrowed by ribs which bend inward with narrowing of the costal angle. Absence of the anterior lumbar curve of the spine results in loss of its shelllike support of the kidneys. These factors are largely congenital and hereditary and are often accompanied by incomplete migration of the gastrointestinal tract. Narrowing of the lower part of the rib cage is added to by tight constriction formerly seen

in women wearing tight corsets now more in men wearing tight belts because suspenders are no longer fashionable. The whole situation is made worse by a stooping slouching posture in children and young people and by failure to exercise the chest in an effort to widen the costal angle. This widening definitely can be accomplished in children when the narrowing is noted and may require removal of upper respiratory obstruction to deep breathing by enlarged tonsils adenoids and nasal obstructions.

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With kinking at the duodenojejunal angle symptoms suggesting obstruction may be produced

*Nephroptosis* may cause pain or dragging feeling in the back on the affected side usually the right. If very low the kidney may cause right lower abdominal pain. With a movable ptotic kidney the ureter may become kinked causing severe right abdominal and back pain. This pain may be mistaken for appendicitis, gallstone colic and intestinal obstruction. With gentle upward pressure on the palpable kidney the kink may be freed with sudden cessation of pain and the passage of a large amount of urine.

*Hepatoptosis* is usually symptomless although dyspeptic symptoms may be present. There are rarely symptoms referable to the hepatic region such as a dragging pain referred to the right shoulder and even colics resembling that from gallstones. Even jaundice has been found ascribed to kinking in the biliary tract. Asthmatic symptoms and slight dyspnea due to downward pull on the diaphragm may occur.

*Splenoptosis* is recognized only by finding the palpable movable low lying organ.

*Coloptosis* usually causes no symptoms although a downward dragging fullness and constipation have at times been attributed to it.

#### PHYSICAL EXAMINATION

In general the patient is of the long narrow asthenic habitus often with adenoid facies, narrow neck and elongated chest showing a small ptosed heart. The costal angle is acute, the upper abdomen narrow, the lower abdomen often protruding with weak flabby musculature. A fluctuating or movable tenth rib is found in over 80 per cent of cases. There may be epigastric pulsation and occasionally tenderness on pressure over the celiac plexus. At times low lying organs may be visible, palpable or percussible with the patient standing. An atonic distended stomach may be seen in the lower abdomen. The liver may be low at times entirely below the costal margin with its upper border determined by percussion depressed below the normal, the fifth intercostal space in the midclavicular line. The kidney may be discovered only by palpation during deep inspiration or may be felt as a movable tumor in the lower abdomen. The distended colon may be seen and percussed as low as the pelvis. It must be remembered that the transverse colon normally may dip down into the pelvis at one time and shortly afterward lie straight across the upper abdomen. It is only when the flexures are low that ptosis may be considered.

#### LABORATORY EXAMINATIONS

Functional tests are of little value. Inflation of the stomach through a gastric tube will define its size and location. Inflation of the colon is of

less value. In general the patient's status as to general nutrition, hemic component and other vital organs is important.

### X RAY EXAMINATION

X ray examination though it can establish the presence of marked ptoses is subject to error unless great care is exercised in determining the distortion caused by distance of the x ray tube from the body and the angle at which the rays strike the organ in question. The appearance of minor degrees of ptosis should be discounted. X ray studies after a barium meal will show the low position of the stomach and may demonstrate kinking at the duodenojejunal angle. Later films may show almost the entire small intestine reposing in the pelvis. In twenty four hours the low colon can be outlined but this is best done by barium enema study. Differentiation of a ptosed from an enlarged liver is difficult. A low lying spleen and kidney may be seen in scout films the latter confirmed by pyelography.

The x ray study is really of most importance in the recognition of diseases of the gastrointestinal tract or elsewhere which may be the cause of symptoms and which might otherwise be attributed to visceroptosis.

### DIAGNOSIS

The diagnosis of visceroptosis as the cause of definite gastrointestinal symptoms should be made with great hesitancy. With careful search lesions not only in the gastrointestinal tract but also in the lungs, heart, kidneys or elsewhere can usually be found as the cause of symptoms. The patient's general body habitus, the nervous symptoms and general undernutrition must all be taken into consideration in making the diagnosis.

### TREATMENT

Prophylaxis is most important. The body habitus can be recognized at birth. The physical development of the child must be undertaken to overcome the narrowing of the chest by proper posture, by proper exercises especially of the chest and abdomen and by avoidance of constricting clothing. Dietary habits and proper rest must be carefully guided. Obstruction to breathing must be corrected often by early removal of tonsils and adenoids. If the condition is seen first in adult life an adequate normal diet of high caloric value to add fat support, proper exercises to develop the abdominal wall especially early after operations or pregnancies and attention to posture in standing, sitting and eating are all important. Abdominal supports of various kinds much used formerly are really a detriment. If they do not fit they make matters worse; if they do fit they weaken the abdominal muscles and discourage the



building up of adequate muscular support by exercise. Rarely a support is needed at best temporarily while general treatment is being carried out.

*Surgical treatment* formerly quite popular need be mentioned only to be condemned. The various forms of suspension and fixation have all been shown to be worse than useless.

### PROGNOSIS

Cure of the body habitus is obviously impossible although in infancy and childhood much can be done with exercises to widen the chest and the costal angle. However with attention to the various measures described with the building up of nutrition and strength patients with *visceroptosis* can be made entirely symptom free.

### Adhesions

There was a time not long ago when any abdominal symptoms not easily explainable on any other organic basis were attributed to "adhesions." Patients were usually willing to accept such a diagnosis and even when no definite locations of the adhesions could be explained to them were ready to undergo repeated exploratory operations in an effort to get relief. With the realization that as a result of each operation more adhesions could be anticipated the public has become more and more reluctant to submit to operations based on such a diagnosis. And yet we know that adhesions may be the cause of distressing symptoms and even of obstruction of the lumen of the gastrointestinal tract. Adhesions may be congenital or acquired.

*Congenital* adhesions include those due to abnormal membranes or veils or to so called bands which may cause deformities, angulations or kinks or actual obstructions. They are found principally in the hepato-duodenal, duodenojejunal and ileocecal regions. Pericolic or Jackson's membranes may cover the proximal or distal colon interfering with motility. Although they frequently cause symptoms in early life they may cause no symptoms and may not be recognized until later.

*Acquired* adhesions may result from four principal causes: (1) *Inflammatory disease* especially that involving or situated close to the serosa. The inflammatory reaction may be due to *intrinsic* disease as from ulcer, biliary tract disease, appendicitis, colitis, diverticulitis, proctitis, tuberculosis or syphilis or to *extrinsic* disease involving or touching the serosa as in mediastinitis, pancreatitis, lymphadenitis, diseases of the genito-urinary tract, peritonitis, psoas infection and others. (2) *Neoplastic disease* especially if near to or involving the serosa. This may also be of intrinsic or extrinsic origin including cancer in the gastrointestinal tract involving other structures by contact or cancer in the neighboring tissues.

involving the gastrointestinal tract (3) *Trauma* likewise of intrinsic origin from ingested foreign bodies or extrinsic from direct or indirect injury to the serosa (4) *Perforation* either complete from any of the causes mentioned above and in turn causing peritoneal adhesions or fistulas or partial walled-off perforation occasioning adhesions to contiguous structures

### SYMPTOMS

The results of adhesions may be only a slight deformity of the regions involved with little or no interference with function and no symptoms. The interference may be sufficient to cause localized pains or spasms, retrostaltic symptoms, hypermotility or intermittent obstructive episodes. In severe cases interference in circulation, obstruction and even gangrene may result. The symptoms resemble those of obstruction due to any cause and occur in the region affected including the esophagus or any part of the gastrointestinal tract.

### PHYSICAL EXAMINATION

In congenital cases other congenital anomalies may be found. In acquired cases the finding of operative scars may lead to a suspicion of adhesions. There is no characteristic finding. Tenderness in the region of a scar is suggestive. With pressure over a distended area gas or liquid may be felt or heard gurgling through a narrowed lumen. At times gentle upward massage may suddenly overcome a kink and allow free passage of contents with sudden relief of symptoms. With severe complications the findings may be those of an "acute abdomen" (see p. 59).

*Proctoscopy* may show narrowing high up in the rectum or sigmoid the nature of which will have to be determined.

### LABORATORY EXAMINATION

Laboratory methods will be of use only to rule out other organic disease.

### X-RAY EXAMINATION

X-ray studies may be of considerable help in making the diagnosis. Congenital bands or membranes may be demonstrable because they may produce narrowing of the lumen or kinks. Esophageal adhesions may not only cause visible narrowing but also may be the cause of traction diverticula (see p. 181). The involved part of the gastrointestinal tract may be deformed and displaced or may show a narrowed lumen or actual obstruction. Kinks may show dilatation behind them which may be relieved if the kinks can be overcome by massage under the fluoroscope. A gallbladder may be adherent to the duodenum or the colon.

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Adhesions from retroperitoneal glands or from an inflamed pancreas or a colonic cancer may deform the gastric outline. The appendix may be adherent to the ileum or cecum with some obstruction proximal to it (Fig 73 p 409). Colonic membranes will fix the colon and smooth its outline. Pelvic adhesions may show annular or extensive narrowing of the rectum or sigmoid (See Figure 78 b page 423). A complete careful study is imperative to determine the original cause if possible. Cancer must be suspected unless it can definitely be ruled out.

### TREATMENT

In the absence of symptoms the finding of adhesions calls for no treatment. With mild symptoms every effort should be made to establish a dietary and hygienic routine which will encourage normal function and relieve symptoms or at least make them intermittent and bearable. *A normal well balanced diet with adequate bulk will overcome many symptoms (see p 21).* Medication is rarely needed although at times sedatives or antispasmodics may be of value to quiet a very neurotic patient temporarily. With mild obstructive symptoms such a regimen should also be tried but if it is not successful operation may be required. For obstruction operation is of course necessary. *The most important factor in treatment is the proper management of the underlying cause or causes.* No patient should be subjected to surgery until all other possible causes of symptoms have been ruled out or adequately treated. Focal infections should be eradicated or treated and the patient brought up to as nearly a normal general status as possible.

### PROGNOSIS

Although at times brilliant results are obtained by surgical measures it must be realized that new adhesions resulting from the operation may cause a recurrence of old or occurrence of new symptoms. Some persons are particularly prone to develop adhesions in spite of the utmost efforts to prevent them.

### Abnormal Pressure

The displacements due to pressure upon parts of the gastrointestinal tract may occur with or may be independent of adhesions. The esophagus can be displaced by changes in size or position of any of the organs in its neighborhood. Normally the esophagus is compressed above by the aorta which when enlarged may cause more compression pushing the esophagus to the right and posteriorly. Below this the compression due to the pulmonary artery may be accentuated in mitral stenosis congenital heart disease emphysema goiter and aneurysm of the artery. Just below this compression made by the left atrium is deepened when the atrium

is enlarged Mediastinal diseases including tuberculosis and cancer may not only cause pressure but also may cause adhesions or a diverticulum

In the abdomen enlarged organs such as the liver gallbladder pancreas spleen kidneys and pelvic organs may cause only pressure with or without adhesions Neoplasms benign or malignant in any location in the abdomen and pelvis may push neighboring parts of the gastrointestinal tract in various directions For instance an enlarged liver may push the stomach duodenum and even the transverse colon downward and to the left An enlarged gallbladder will often deeply indent the duodenal cap and an enlarged spleen will cause a large defect of the greater curvature of the stomach Compression by an enlarged pancreas is often diagnostic (see p 606) An enlarged uterus or distended bladder can push the small intestine upward the rectum and sigmoid to the left An enlarged kidney may push the colon forward or to one side Retroperitoneal masses may push the stomach duodenal cap small intestine and transverse colon forward

### SYMPTOMS

There may be no specific symptoms or there may be symptoms related to dysfunction as a result of displacement These might include any syndrome from retrostalsis to real obstructive symptoms The most prominent symptoms are usually those of the underlying cause

### PHYSICAL EXAMINATION

Physical examination must be thorough and may disclose evidences of the cardiac pulmonary aortic abdominal or pelvic condition causing the displacement There may be no more than a suspicion that the gastrointestinal tract is being compressed except that abnormal distention may point to partial or complete obstruction

### LABORATORY EXAMINATION

Laboratory aids may be helpful in discovering the primary condition or the effects of compression anorexia food deficiencies and dehydration

### X RAY EXAMINATION

X ray examination will usually demonstrate the condition A scout film may disclose enlargement of solid organs with the effect on a gas containing gastrointestinal tract (See Figure 92 page 479) A gastrointestinal series and a barium enema will show the pressure defects and the displacements or obstruction (Fig 9) The special x ray studies for cardiac pulmonary urological hepatic and pelvic disease may be required before a complete diagnosis can be made as to the cause of the pressure and displacement



*Figure 9* Pressure of enlarged liver (cholangioma) on stomach. Stomach pushed to left and downward by the enlarged liver. (Used through the courtesy of Herbert Friedman M.D.)

### TREATMENT

Usually the treatment of the underlying cause of the pressure is the most important consideration. There is no other special treatment for the pressure. The gastrointestinal tract should be kept functioning as normally as possible by encouraging an adequate well balanced diet with between meal feedings to maintain normal peristalsis and to prevent biliary stasis (see p. 21).

### PROGNOSIS

The results of treatment depend upon the ability to correct the underlying cause. When enlarged organs can be reduced in size, circulatory defects corrected, pulmonary complications relieved and neoplasms removed, it can be expected that the gastrointestinal tract will be restored to normal function.

### Herniation

The displacement of the parts of the gastrointestinal tract produced by hernias is usually not extensive but may be of serious import. Unfortunately, although hernias are not at all uncommon, they are too frequently overlooked and neglected. There is still too great a mortality from strangulated hernias which are usually the result of neglect.

Herniation may take place in any part of the gastrointestinal tract. Hernias are usually classified as internal and external although the line of demarcation is not well defined. They may be congenital or acquired.

### INTERNAL HERNIAS

*Diaphragmatic* hernias are described in the section on the Esophagus (p 184). They may be small or large, congenital or traumatic. They may occur through either side of the diaphragm, more on the left, or through the esophageal hiatus. They may contain only parts of the stomach and lower end of the esophagus or the whole stomach, and in severe cases also all other abdominal organs, only the rectum and genitourinary tract remaining in normal position. Treatment may be medical, surgical or palliative, with varying results.

*Abdominal* internal hernias may result from a section of intestine or omentum getting caught under congenital bands or membranes, under acquired adhesions between abdominal organs, or as a result of inflammation or trauma, often from operations. Small intestinal loops may slip through a poorly closed aperture in the transverse mesocolon after gastroenterostomy, or may slip behind the jejunum at its anastomosis with the stomach. Internal hernias may involve the duodenum, which is surrounded by fossae into which a loop may protrude. Hernias may also occur through the foramen of Winslow, into the intersigmoid fossa, through the broad ligament, or may result from various degrees of nonrotation. The large intestine is rarely herniated.

### COMPLICATIONS

When the hernia is not reduced spontaneously or at operation, more or less obstruction is usual. It may become complete and cause gangrene.

### SYMPTOMS

There are no definite symptoms suggesting internal hernia. The symptoms are usually those of an intestinal obstruction, partial or complete (see p 67).

### PHYSICAL EXAMINATION

There is no characteristic physical finding. Abnormal distention of the left side of the abdomen is suspicious. With obstruction, the findings of an acute intestinal obstruction are present.

### X RAYS

A scout film may show abnormal accumulation of gas-filled intestinal coils with distention above. Films following a barium meal may show more clearly the coils of intestine apparently enclosed in a definite space (see Fig 10). Distention above this will depend upon the degree of the obstruction. Without obstruction, the barium will pass beyond in a



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Ventral hernias may be due to a localized weakness in any part of the abdominal wall and may be large or small congenital or acquired as a result of trauma or operation (incisional hernias). Epigastric hernias usually occur through a weak spot in the linea alba. They may be as small as a split pea or may be large containing omentum or intestine and even stomach. Diastasis of the recti muscles may also be considered among ventral hernias containing as it often does a large part of the abdominal contents. More rarely herniation may occur through the obturator canal or membrane below the rimus of the pubes through the edge of the quadratus lumborum the sacrosacral foramen or between the vagina and ischial ramus or the vagina or prostate and rectum.

### SYMPTOMS

External hernias may produce no symptoms until complications have developed. They may cause localized pain at the site of protrusion or peristaltic pain above it if the bowel is partially occluded. Such symptoms may disappear when the patient lies down and the hernia is reduced. The patient may feel the protrusion and reduce it himself by recumbency and manipulation without seeking medical advice for years. He may attempt to prevent protrusion by home made or advertised appliances. He may be afraid to see a doctor because he fears that an operation will be advised. Applying to a doctor for other care he may not mention the protrusion and if he is examined only lying down it may be overlooked.

### COMPLICATIONS

Intestine may become incarcerated in a hernial sac and may resist all efforts at reduction even though no obstruction of the lumen may be produced. With obstruction all the symptoms of intestinal obstruction will be present. Occasionally patients not specifically examined for herniation at the various locations mentioned may go on to strangulation and gangrene of the gut and undergo operation before the real site of the obstruction is recognized.

### DIAGNOSIS

The history may be deceptive the pain is not characteristic and the protrusion may not be mentioned or may be concealed.

### PHYSICAL EXAMINATION

Physical examination is most important. No physical examination is complete without a search for possible hernias. With the patient examined only lying down the hernias are almost always missed unless perchance their contents are not reduced in the supine position. The areas of possible protrusion should be examined in various positions standing, stooping and, particularly in the case of ventral hernia when the patient





Figure 10 Internal hernia (1) Stomach (2) small intestine in large hernial sac (3) small intestine beyond hernia

normal manner. The stomach and colon may be displaced. There may be evidences of nonrotation.

#### TREATMENT

The only cure for internal hernia is surgical operation. Undoubtedly some persons go through life with herniated loops of bowel not tightly compressed and not obstructed without ever having symptoms. When symptoms are present and the evidences of herniation are found, surgery is indicated.

#### EXTERNAL HERNIAS

*External hernias* are those which occur through congenital or acquired canals or openings in the abdominal wall. The contents of such hernias may consist only of protruded inner layers of the abdominal wall or peritoneal or omental fat or of one or more abdominal organs. The congenital type includes those occurring at the inguinal or femoral rings or at the umbilicus. There may be only weakness at these points (potential hernias) with an impulse felt on coughing. After a sudden strain or lifting or after laparotomy, actual protrusion may occur on standing, usually reduced on lying supine. Large hernias which may extend down into the scrotum may contain many feet of intestine, usually only small but rarely also large as well as the appendix.

second most frequent cause of death exceeded only by heart disease. Nearly one quarter of a million deaths from cancer can be expected each year of which nearly 40 per cent or 100 000 will be due to cancer of the digestive tract. Whether the increase in cancer is due to the increased average age of our population permitting more people to attain the so-called cancer age or whether it is due to better diagnosis and more accurate reporting of cancer deaths is only of academic interest. The important fact is that with gastrointestinal cancer patients living approximately one and one-half years after the onset of symptoms there must be more than 150 000 patients with gastrointestinal cancer living in this country. In spite of the fact that the various cancer societies and the Federal Government have been spending a great deal of money and energy in teaching the public the importance of early diagnosis to effect a cure in cancer in spite of the expansion of facilities for the screening out of cancer cases in spite of more and more cancer courses and conferences for the medical profession it is sad but true that the majority of patients with gastrointestinal cancer are still reaching the surgeon too late for cure. Unfortunately until the tremendous volume of research work being done results in the production of a medical cure for cancer surgical extirpation will remain the only possible cure and getting the patient to the surgeon promptly must be the aim of every one who sees a cancer case.

Early diagnosis the most important factor in the cure of cancer has been hampered instead of aided by much that has been written about gastrointestinal cancer. Most textbooks and articles give too much space to the description of the findings in advanced cancer and not enough to real early diagnosis. If by early diagnosis is meant recognition of cancer in time for cure it is a fact that an early diagnosis of gastrointestinal cancer is rarely made as evidenced by the high mortality even when the most modern surgical methods are resorted to. The failure of early diagnosis is often blamed upon the patients who are often too afraid to face the possibility of such a diagnosis it is claimed that they do not pay attention to early mild symptoms and that they refuse to have a complete gastrointestinal study even if offered to them free. However the doctors themselves are just as frequently at fault in that they do not bother about taking a complete history when patients present only mild gastrointestinal symptoms in that they are prone to include gastrointestinal symptoms in general among psychosomatic manifestations and in that they do not in the presence of symptoms insist on a complete gastrointestinal study to rule out cancer. The screening of all the fifty million persons in this country past forty years of age for gastrointestinal cancer even though no symptoms were present would not be practicable. Facilities and personnel for a complete gastrointestinal survey not only are not

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strains the abdominal wall on rising himself from the bed to the sitting position. Reduction by posture or manipulation clinches the diagnosis. Percussion, transillumination or the hearing of a gurgling sound with reduction may establish the presence of intestine in the sac.

### X RAY EXAMINATION

Occasionally an x ray study will disclose a hernia which has been missed on physical examination. Small intestine extending below the normal lower limit can be seen in the x ray films lying in an inguinal hernia even in the scrotum. Films in lateral and oblique positions may be needed to show protrusions elsewhere.

### TREATMENT

The treatment for external hernias is surgical. Strangulated hernias will of course require emergency operation. Incarcerated hernias should always be operated upon to prevent strangulation. In reducible or symptom free hernias in patients who for some reason will not consent to operation or are in physical condition prohibiting its use, trusses may be of value but mostly in the inguinal or femoral hernias. Ventral hernias are poorly confined by abdominal supports with inserted pads. Injection of sclerosing agents in the inguinal region is at times successful.

### PROGNOSIS

Although surgical techniques are constantly improving, there is always the possibility of recurrence after operation. At times two or three operations have been performed before a cure was effected. Patients with large ventral hernias or diastases, while somewhat incapacitated, may never suffer complications.

## Gastrointestinal Cancer

Although cancer of each organ in the gastrointestinal tract will be discussed in the chapters devoted to each organ, it is distinctly worthwhile to discuss gastrointestinal cancer in general.

### INCIDENCE

The incidence of and the death rate from cancer in this country have about doubled in the past twenty years and cancer has become the

ally through the veins. The liver is most frequently involved although metastases to lymph nodes in the pelvis the mediastinum alongside the spine and to the left supraclavicular node known as "Virchow's gland" are more or less frequent. Distant metastases to the bones lung or brain occur rarely.

#### TYPES

Carcinoma is the type of malignant neoplasm found in over 90 per cent of gastrointestinal malignancies. The type of cancer encountered depends somewhat upon the location of the lesion.

In the *esophagus* squamous cell carcinoma is most common. Adenocarcinoma may occur as a result of upward extension of a gastric cancer or as a result of malignant change in ectopic gastric mucosa in the esophageal wall. Both are rare as are malignant lymphomas lymphosarcoma and Hodgkin's disease, this last being the most common of these cancers.

In the *stomach* adenocarcinoma is the rule with colloid medullary papillary and scirrhous (linitis plastica) varieties occurring at times. The lymphomas are rare lymphosarcoma leiomyosarcoma and Hodgkin's being the ones usually encountered with melano-fibro- and angiosarcoma most rare.

In the *intestines* the malignancies are usually of the same types as in the stomach. In the *rectum* basal cell carcinomas are most common although scirrhous and villous adenocarcinomas may occur.

In the *liver* primary carcinomas are rare and occur as liver cell carcinomas called hepatomas bile-duct cell carcinomas called cholangiomas and atypical or mixed types. Secondary carcinomas are most frequent (a ratio of 5:1 of primary) and lymphomas are more rare.

The *gallbladder* and *bile ducts* are rarely the seat of adenocarcinoma and sarcoma as myosarcoma occurs even more rarely.

In the *pancreas* malignant neoplasms also rarely occur. Islet cell adenocarcinoma and ampullary carcinoma as well as sarcoma and malignant cysts are the ones encountered.

*Carcinoids* although not generally considered among the malignant tumors occur mainly in the intestines appendix and rectum. They are rare and may occur as single or multiple tumors. They are difficult to differentiate from cancers in fact they may be precursors of carcinoma. In the small intestine they frequently metastasize early and are almost invariably fatal although death may not follow for as long as ten years. In the rectum metastases have also been seen extending up to the liver.

*Leukemia* though not showing definite lesions of the gastrointestinal tract may cause retrostaltic symptoms possibly due to splenomegaly.

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available but even if available are too expensive. The screening must therefore be confined to patients who have gastrointestinal symptoms especially to those giving a family history of cancer.

### ETIOLOGY

Although the cause of cancer has not yet been determined there have been accumulated certain facts about this disease which are of use in diagnosis or treatment. We know that although gastrointestinal cancer may occur at any age it is most common after forty. Observations on heredity in mice and human beings have indicated that susceptibility to cancer is a recessive resistance to it a dominant characteristic. We know that irritation plays a part in causing cancer and its frequent though not constant occurrence at points of greatest irritation or activity in the gastrointestinal tract and in patients who have had chronic irritation from irritating foods or drugs or from allergy seems to support this observation. We know that some benign tumors tend to undergo cancerous degeneration and we find this true of gastrointestinal adenoma papilloma leiomyoma and others. It has been demonstrated that carcinoids are frequently precursors of signet ring adenocarcinoma. We know that at times chronic lesions called precancerous tend to become malignant as in gastritis colitis diverticulitis cholelithiasis and hepatic cirrhosis. Recently collagen diseases have been also suggested as precursors of cancer. We know that some agents known as carcinogens are prone to cause cancer and ingestion of excessively hot liquids or foods or of various aniline dyes and other non nutritive additives now used extensively in our foodstuffs has been postulated as belonging in this category. Gastrointestinal cancer also occurs as a direct extension from contiguous organs or as a result of metastases the liver being particularly the target for metastatic involvement from distant organs.

Carcinoma of the gastrointestinal tract usually occurs in three forms (1) a growth extending into the lumen producing a fungating mass (2) a growth infiltrating the wall usually tending to ulcerate and (3) less commonly a cancer extending along the wall either in the deeper layers producing limitis plastica or in the mucosa or submucosa producing the superficial type. Malignant lymphomas are rarely found but when present are usually in the stomach and small intestine seldom in the colon. Sarcoma rarely starts in the wall of the gastrointestinal tract occurs occasionally as a result of sarcomatous change in a leiomyoma but is mainly due to pressure and then infiltration from a sarcoma originating in the vicinity often from retroperitoneal lymph nodes. Hodgkins disease also spreads from these or thoracic lymph nodes.

The spread of gastrointestinal cancer is either by direct extension or contact or by metastases mostly through the lymphatics but occasion

ally through the veins. The liver is most frequently involved although metastases to lymph nodes in the pelvis, the mediastinum, alongside the spine and to the left supraclavicular node known as "Virchow's gland" are more or less frequent. Distant metastases to the bones, lung or brain occur rarely.

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## SYMPTOMS

In discussing gastrointestinal malignancy the symptoms usually described are those found in advanced cases so that if we aim to arrive at an early diagnosis they had best be forgotten. They include the progressive loss of weight and strength the marked cachexia the toxemia the fever and finally the development of a palpable mass or enlarged lymph nodes or of complications such as obstruction hemorrhage or perforation with resultant pain vomiting diarrhea hematemesis melena or an acute abdominal calamity. By the time these symptoms appear most cases have become incurable.

The *early symptoms* are most important although frequently it is true that no noticeable symptoms occur until complications have developed to call attention to the condition. At times typical peptic ulcer symptoms are present. Absence of previous ulcer symptoms should lead to a suspicion of cancer. In most cases however the symptoms at first are mild and develop insidiously.

In *esophageal* cancer dysphagia occurs early and substernal pain may precede it. The tumor even though small acts as a foreign body an irritant modifying the secretion and the motility of the organ in which it is growing. The early symptoms of *upper gastrointestinal* cancer are usually indefinite retrostaltic in type described by the patient as mild indigestion epigastric fullness or unrest desire to belch sour eructation or regurgitation nausea rarely vomiting anorexia with occasionally a disgust for food. In *biliary tract* cancer jaundice may be an early symptom. In the *lower gastrointestinal* tract there is increased peristalsis with mild cramplike feelings distention flatulence and mild diarrheal spells. This tendency to diarrhea is usually considered by the patient to be a favorable sign even though mucus may be noted in the stool. In *rectal* or *sigmoid* cancer hemorrhoids may develop as a result of interference with venous return and a little bleeding will be attributed to piles causing no worry. Decreased intake of food or increasing diarrhea may cause general weakness loss of weight dizziness and pallor and the patient may take tonics iron or vitamins for them. At this stage there may still be hope of cure but the more severe symptoms are usually awaited before medical advice is sought.

Occasionally the *onset* of symptoms is sudden. With no previous symptoms of any gastrointestinal complaint there may occur a sudden hematemesis and melena or evidences of an acute abdomen due to perforation or obstruction. The patient may consult the physician about a swollen gland above the clavicle or about a hard tumor in the abdomen or there may be the development of a severe painful bloody diarrhea resembling ulcerative colitis.

## DIAGNOSIS

The history is most important for an early diagnosis. The most characteristic history is one of no previous gastrointestinal complaints and the gradual onset and persistent progression of some of the symptoms noted above. In malignant degeneration of a previously existing disease there is a development of new symptoms in addition to those previously present. It is upon the suspicion aroused by these two types of history that early diagnosis depends.

## PHYSICAL EXAMINATION

Physical examination early may show no abnormal findings except perhaps a little pallor or evidence of slight loss of weight. By the time an abdominal mass can be made out or lymph nodes or nodules in the axilla or neck become palpable the hope of cure has become questionable. A rectal digital examination may however disclose a small hard nodule and proctoscopy may demonstrate a small polypoid mass which bleeds easily.

## LABORATORY FINDINGS

The blood may show a mild secondary anemia. Leukocytosis or rapid sedimentation rate may be a late finding when there has been much sloughing, ulceration or secondary infection. Hyperglycemia and glycosuria may be present in pancreatic cancer and increased serum alkaline phosphatase and prolonged prothrombin time in hepatic cancer. Persistent findings of occult blood in the stools if all other causes can be ruled out indicates persistent ulceration such as usually is associated with cancer. Visible bright red blood occurs fairly early in rectal cancer. The passage of a stomach tube may be delayed in esophageal cancer and may show bright blood on its tip and cancer cells on microscopic examination. Fractional gastric analysis may show normal or abnormal acid secretion but the finding of a little bright blood in the content should occasion a suspicion of neoplasm.

*Cytologic studies* of vomitus or gastric content or rectal and colonic discharges especially of specimens obtained by means of brushes or inflatable balloons covered with an abrasive silk mesh or by means of other contrivances to remove fresh gastric, rectal or colonic contents may disclose cancer cells. Biopsy through an esophagoscope, gastroscope or proctoscope may clinch the diagnosis.

*Peritoneoscopy* not only permits direct observation of abdominal organs and of tumors and metastases but also permits removal of specimens for biopsy. It is particularly valuable when a mass can be felt. If the biopsy should disclose a lymphoma it would be of value in suggesting radiation therapy rather than operation.



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Punch biopsy may fail to obtain specimens of a cancer of the liver or elsewhere and may produce a false sense of security. Repeated punch biopsies increase the risk inherent in the procedure. Removal of palpable lymph nodes may disclose a cancer which would then be incurable. Cancer diagnostic tests have been devised but as yet none is reliable.

### X RAY EXAMINATION

X rays are the most valuable aid to early diagnosis when no biopsy is possible but require great experience and most scrupulous care to bring out small early lesions. The x ray study must be made in different positions and at different angles with varying amounts of contrast medium by means of fluoroscopy and films. A complete gastrointestinal study with barium enema and gallbladder studies is imperative. A small cancer involving the mucosa may at first reveal a small area of stiffening through which peristaltic waves will not pass and as a tumor develops will show a widening of the stiffened area and a defect where the tumor protrudes into the lumen. When it grows larger the irregular outline of a polypoid mass may be seen and with sloughing an irregular defect with areas of ulceration may develop.

In the esophagus the growing mass will cause fairly early narrowing of the lumen. In the gastric fundus the tumor may grow to large size and go unnoticed unless supine films are taken to fill up the fundus. Cardiospasm may occur early even without involvement of the cardia. Later the growth may extend to the cardiac orifice and cause esophageal obstruction or may involve more or less of the remainder of the stomach. In the pyloric antrum more or less obstruction may occur fairly early. An unusual form of gastric cancer usually a scirrhus carcinoma may resemble a benign ulcer with a defect and crater. This can be differentiated from a simple ulcer only by repeated observation in three or four weeks when persistence or increase in size will indicate a cancer. In the small intestine the lumen is soon encroached upon and signs of obstruction with dilatation behind a narrow point will be seen. In the cecum ascending and transverse colon a small tumor may easily be overlooked but irritability with increased peristalsis should occasion a careful mucosal study with air inflation since obstruction is apt to be late. In the descending colon and sigmoid even small tumors are frequently demonstrable as small defects or polyps or as annular constrictions which tend to obstruct early. Rectal lesions are difficult to discover and are usually demonstrated by x ray only after the digital or endoscopic examination has discovered them. External pressure and involvement are seen when retroperitoneal masses (usually lymphomas) and hepatic pancreatic bowel or even renal splenic or pelvic tumors press into any point of the

gastrointestinal tube. This pressure will produce fairly characteristic defects which can be demonstrated not to involve the mucosa early but perhaps to involve it later. Liver tumors aside from causing hepatic enlargement and esophageal varices may at times present a differentiable tumor which may be further brought out by hepatography or angiography. Cancers of the biliary tract and pancreas are recognized by their pressure effects or actual infiltration of stomach or duodenum. By x ray alone it is usually impossible to differentiate the various malignant neoplasms. The x ray findings are described in more detail in the chapters on cancer of each organ.

### TREATMENT

By cure of any disease we usually mean that complete disappearance of the disease has resulted either from natural causes or due to specific treatment. Until a therapeutic agent is found which will assure the complete annihilation of all cancer tissue in the body, we cannot rightly speak of cure of cancer although of course if a cancer is definitely localized and can be totally removed it could be considered cured. In gastrointestinal cancer there is as yet no curative agent and with the insidious spread of the cancer cells often to distant locations the term "cure" is hardly applicable. Even with thorough extirpation of the primary growth and all visible or suspected metastases the surgeon can never be sure of cure. So far surgical treatment is the only method by which a possible cure can be effected.

*Radiation* by x ray, radium or newer radioactive elements such as Cobalt-60 though it may at times cause a dissolution of superficial cancers has not proved to be of more than temporary palliative value in gastrointestinal carcinoma and its use as a prophylactic before or after operation has also been of little value. Sarcoma in general is somewhat amenable to radiation therapy but real cures are rare. Hodgkin's disease is not cured in this manner. The radioisotope of phosphorus has been successful in some cases of reticulum cell sarcoma.

*Fulguration* or electrodesiccation with the so called diathermy knife of considerable value in superficial cancer has been of proved value only in some cases of rectal cancer.

*Hormonal* and *endocrine* treatments and the use of *chemicals* such as nitrogen mustard and the "antiblastic" krebiozen combined with other methods of treatment are probably of some value but are not curative in themselves. Nitrogen mustard with or without radiotherapy besides being of value in malignant lymphomas is being used immediately after excision of cancer to "mop up" stray cancer cells. It is injected intravenously or intraperitoneally. Mycotic and staphylococcal infections may follow chemical therapy.

## Surgical Treatment

Surgical treatment the only present method of possible cure is being constantly improved and today many patients previously considered inoperable are being subjected to more and more extensive operations so that in many cases it appears as if the patient were being dissected away from his tumor rather than vice versa.

*Preparation for Operation* The success of any operation is dependent upon adequate preparation. Unfortunately many clinicians as well as surgeons are in a great hurry to operate upon suspected cancer patients rush them into a hospital and insist upon immediate operation. Undoubtedly many lives are lost as a result of this haste. In the absence of obstruction whether the patient is operated upon a few weeks sooner or later has little if any effect on the curability of the lesion but that much time spent in preparing the patient for operation will certainly permit more complete surgical procedures and definitely reduce the mortality not only from the operation but also from a spread which may be overlooked in hasty procedures.

In preparation for operation there must be (1) a *complete evaluation* of the patient's ability to withstand an operation including studies of the respiratory, cardiovascular and urinary systems and careful treatment of any disease conditions discovered.

2 Any *abnormality discovered* in the complete gastrointestinal x-ray study which is amenable to treatment should be adequately treated.

3 Any *focal infections* in the mouth, nose, throat, pelvis or rectum should be removed or actively treated in order to avoid postoperative complications from inhalation, absorption or vascular dissemination.

4 *Psychological preparation* should consist in allaying the fears of patient and family, encouraging their cooperation in preparing for operation and instilling the hope of cure. In general I do not believe that a patient should be told that a cancer is present but at times it is necessary to explain that if operation is not performed the condition might become cancerous. A plausible explanation for the symptoms must be given such diagnoses as ulcer, gallbladder trouble, colitis or hemorrhoids being most readily accepted.

5 *Exercise* is important to prevent postoperative atelectasis and phlebitis. Whenever at all possible the patient should be encouraged to walk, do simple setting up and deep breathing exercises preferably in the fresh air. Rest periods should also be insisted upon. Even if he is confined to bed some exercises supplemented by massage are of value.

6 *Nutrition and fluid balance* are most important. If there is no obstruction and the patient can swallow easily a nutritious well balanced diet with added vitamins and minerals should be prescribed (see p 21). Even only a week or two of such a diet will do much toward promoting normal convalescence. In the presence of ulceration an ulcer

type of diet would be useful. If there is a partial obstruction the diet will need to be of liquid or thin consistency and may have to be supplemented by parenteral feedings or transfusions in order to attain fluid and electrolyte balance. While exact determination of such balance is of value opportunity to perform frequent accurate determinations is not often available. The clinician must depend largely upon observation of dehydration of mucous membranes as a guide to fluid administration and the avoidance of excessive saline infusions in order to prevent hypokalemia. In general balanced saline infusions such as Ringer's solution are preferable to simple sodium chloride.

Liquid feedings should supply a balanced ration. There are now available canned powder preparations which when dissolved in water will provide not only normal proportions of protein fat and carbohydrate but also all necessary minerals and vitamins to supply a normal food intake. These may be taken by mouth or through a nasal tube. Various preparations are now available also for parenteral administration. Many persons dislike the taste of the prepared mixtures and prefer such a sweet liquid as I have used for many years. Directions for this diet may be given to the patients as follows:

### Liquid Diet

Put a clean empty milk bottle place 3 ounces of heavy cream and 3 ounces of dextrose fill with homogenized milk keep this in the refrigerator.

At each feeding dissolve a rounded teaspoonful of powdered gelatin in an ounce or two of the warmed mixture and add to the remainder taking the chill off the mixture and giving it a "body." Occasionally when the patient objects to the flavor a little vanilla flavor may be added or the doctor may order a little whiskey. If eggs are to be added they are beaten up and mixed in with the feeding. Exact dosages must be obtained from the doctor.

This mixture contains over 1200 calories per liter with 80 gm of protein 100 gm of carbohydrates and 65 gm of fat. The gelatin besides providing protein prevents the formation of large curds the resulting fine curds being so small that they can be aspirated through a fine stomach tube. When more protein is required two or three raw eggs per day can be added and the addition of alcohol either pure (20 to 30 cc per day) or in the form of whiskey or brandy (40 to 60 cc per day) will increase the caloric value and produce mild sedation. The amount of feedings usually given varies from 4 to 8 ounces at intervals of two to three hours depending on the condition.

After several days of these feedings some patients will have loose stools which can be controlled by reducing the sugar content or by giving alternate feedings of cereals fruit juices raw eggs and vegetables reduced to a liquid in a blender. In case of allergy to milk, substitutes such as soy bean preparations may be used. In any case mixed vitamins and minerals should be provided either by dissolving preparations of these elements in the mixture or giving them separately using extra

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doses of such vitamins as the patient's condition may require when there is evidence of specific deficiencies

*Laxatives* should be avoided as much as possible Mineral oil in 1/2 ounce doses at night and as rectal retention enemas of 5 or 6 ounces are usually satisfactory although at times a cleansing enema may be indicated

In case of *emergency* such as obstruction or suspected perforation preparation for the operation must of necessity be brief Transfusions are indicated and the patient can be nourished by parenteral injections of dextrose amino acids electrolytes and alcohol in an attempt to approach a normal fluid and electrolyte balance Antibiotics are also given parenterally

*Immediate* preparation for operation is left to the surgeon and anesthesiologist In general most surgeons prefer not to have purgation before operation although in some types of operation this is considered imperative Antibiotics or sulfonamides are usually given for a few days before operation if possible The anesthesiologist orders preoperative sedation as deemed necessary

*Scope of Operations* The surgical treatment of gastrointestinal cancer has undergone marked changes since the advent of antibiotics the newer anesthetics and massive continuous transfusions Even in the case of fairly early lesions surgeons today prefer to perform much more radical operations in order to make sure that no metastases have been overlooked In esophageal cancer not only can large sections of the esophagus be removed but also portions of involved pleura can be resected and the rent closed The proximal end of the stomach may be mobilized so that it can be brought up and anastomosed with the esophagus even as high as the aortic bifurcation In gastric cancer not only a part but all of the stomach can be removed and in esophagojejunostomy done When much of the esophagus is also involved the jejunum can be brought up into the chest In cancer of the pyloric region biliary tract pancreas and duodenum all these organs and even parts of the colon and omentum are being removed with anastomoses between stomach and hepatic and pancreatic ducts and the jejunum In colonic cancer the involved segment and any adjacent involved organs are excised the most extensive operation of this type consisting in removal of the rectum sigmoid pelvic organs and bladder with the ureters implanted in that part of the descending colon which is above the permanent colostomy Whereis formerly cancer of the liver was considered inoperable today extensive resections are possible Though the mortality of these extensive operations has been steadily reduced and the number of five year survivals has been slightly increased it cannot be said that many of the poor victims have been benefited to the extent that they could enjoy life It is hard to con

ceive of any more uncomfortable situation than to have a permanent colostomy through which both urine and feces are expelled

*Postoperative Care* Postoperative care of gastrointestinal cases in the past unfortunately was governed largely by fads and fancies. It should be left to clinicians with special training in gastroenterology. A thorough knowledge of nutrition and gastrointestinal and cardiovascular physiology would have avoided many of the past and present errors in postoperative care. Looking backward it would have avoided the evils of the excessive use of the Murphy drip of hypotonic tap water of the overwhelming waterlogging and the depletion of blood potassium and vitamins of patients by the infusion of excessive amounts of sodium chloride of the gastrointestinal atony and weakened respiration from overdosing of patients with opiates and other narcotics of the further gastrointestinal atony to the point of meteorism as a result of starvation of the depletion of chlorides bile salts and pancreatic juice from the pernicious continuous suction of the tendency to atelectasis and venous stasis as a result of prolonged bed rest

Today it is realized that early feeding and early ambulation are most important. Most surgeons have enough faith in the strength of their wounds to let the patient out of bed a day or two after operation but they appear not to trust their anastomotic suture lines sufficiently to allow real feedings until several days later. Early feeding encourages normal secretion and normal peristalsis avoiding postoperative distention and stasis. Liquid feedings such as described under preoperative care are admirable for this purpose. While the abdomen was open and the patient under an anesthetic I have seen active peristalsis initiated upon slight distention of the stomach or small intestine with such a liquid mixture through a tube. Rarely do we encounter any postoperative discomfort such as is supposed to be avoided by the unphysiologic postoperative suction. Added vitamins and minerals must not be neglected.

After three or four days of the liquid feedings additions are made to every other feeding starting with cereals puddings ice cream poached eggs and baked potato in increasing quantities until in a week or ten days the patient is on an ulcer diet (see p 267). To this in turn are added vegetables fruits and finally meats until in a month or so a full well balanced diet can be taken. Under such postoperative care I have never seen a patient with so-called dumping symptoms.

Gradually increasing exercise including particularly abdominal exercises as well as mild setting up exercises and walking preferably in the fresh air will tone up not only the gastrointestinal tract but also the body as a whole greatly shortening the time till the patient can get back to work. Proper care of colostomies and ileostomies will do much to alleviate the discomfort experienced by patients condemned to live with them (see p 394). Care of the rectum after operation should include

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oil retention enemias soothing suppositories and when bleeding occurs hot retention enemias of 10 per cent gelatin solution 5 or 6 ounces at 110 to 115° F (see p 47)

### Palliative Treatment

Palliative treatment is unfortunately still an important part of the treatment of gastrointestinal cancer. In incurable cancer such *palliative operations* as gastroenterostomy, enteroenterostomy, colocolostomy or cholecystoenterostomy may give a patient months of comfort. It is questionable, however, whether establishment of an external fistula such as a gastrostomy or an enterostomy is justifiable. The discomfort, the physical and psychological distress, the incapacity for useful work attending such openings would make it seem more humane to let the disease take its natural course unless a few weeks or months of prolonged life will permit attention to personal matters of great moment. The same is true to a lesser degree of palliative ileostomy or colostomy. I have seen patients live with *unbearable discomfort for a year or more after such operations*, especially in old persons in whom the cancer may grow more slowly.

Medical palliative care is of great importance in making the patient more comfortable during his few remaining weeks or months. From the psychological standpoint I believe that patients prefer not to know they have cancer or that it is incurable. If they have had a palliative operation it can be described as having achieved its purpose, but that permanent relief of all symptoms will take a long time. It is often a help to set the time of complete recovery about the time the patient can be expected to die. It is also of help to predict that before recovery certain symptoms such as vomiting or obstipation may occur so that the impact when they do occur will not be too great. The family must be instructed to try to keep the patient cheerful, to bring in news from the outside and perhaps to make plans with the patient for activities or trips when he gets well. Sedation, most important in the later stages, can often be postponed by using aspirin, phenacetin or other analgesics to relieve pain, barbiturates, bromides or other tranquilizers to encourage sleep and Chlorpromazine for the relief of nausea and vomiting. As soon as necessary sedatives starting with codeine and continuing with morphine orally or parenterally or opium in suppositories should be used in constantly increasing doses, not only to relieve symptoms but also to dull the patient's mind to the apparent incurability of his disease. There is little if any advantage in using the newer, more expensive antispasmodics and narcotics. I have had patients who received as high as 20 or more grains of morphine per day hypodermically yet remained conscious and even cheerful.

*Feeding is often a difficult problem. Anorexia may at times be allevi-*

ated by the use of bitter tonics or a cocktail before eating. The type of food whether liquid, soft or normal must be guided by the location and effect of the growth. In obstruction a liquid or nonresidue diet would be indicated but when a short circuiting operation has been performed a normal diet can be taken for a time. The patient will appreciate it if allowed to eat foods he likes particularly but tends to become suspicious unless some restrictions are made.

Other palliative measures would include paracentesis for ascites or pleural effusions. Recently radioactive strontium ( $^{90}\text{Sr}$ ) 30 to 40 micrograms injected into the serous cavities at the time of paracentesis has been found to prevent reformation of fluid in half of the cases. Colloidal gold was formerly used as was radiation to the pleura. Oxygen inhalations for marked dyspnea and attention to cardiovascular renal complications are also indicated.

### PROGNOSIS

In spite of improvements in early diagnosis and treatment in spite of the daring surgical procedures now possible in spite of increasingly better results in colonic surgery the prognosis in gastrointestinal cancer is still not good. Until some general cure is discovered we must be content with being able to prolong the life of the cancer patient.

## Gastrointestinal Psychosomatic Disturbances

The effects of emotions on gastrointestinal function have been recognized since time immemorial. Normal psychic stimuli have long been recognized as valuable in promoting normal function. As examples are the effect of the sight and smell of food on salivary and gastric secretions and the thought of defecation inducing a normal bowel movement. Abnormal stimuli have also been known to cause more or less violent effects such as the diarrheal stool occurring with fear or apprehension, the anorexia or dysphagia with grief, the nausea and vomiting with disgust. Whether prolonged or repeated abnormal emotional stimuli can produce chronic symptoms or even actual organic disease is open to question. Upon the supposition that they can do so have been based the psychiatrists' teachings in the field of psychosomatic medicine.

At first it was contended merely that many gastrointestinal symptoms



were due to psychic stimuli but in more recent years actual somatic diseases such as peptic ulcer and ulcerative colitis have been taken over by the psychiatrists with the contention that they are definitely the result of previous or present psychic trauma. The interesting scientific studies of Selve have demonstrated that stress whether psychological (emotional) or physical (trauma including surgical operations, infections and their sequelae) has a definite effect on the endocrine system. Instead of accepting this action as evidence of a physical (glandular) cause for the phenomena observed, instead of stimulating a search for possible hidden physical stresses such as chronic infections or other diseases, psychic stress alone has been emphasized and Selve's observations have been considered a confirmation of the theory of mind over matter. This has hastened the development of the elaborate system of psychosomatic medicine with which we have to contend today.

Under this system it is now a fact that many of its practitioners feel that an exact diagnosis of a gastrointestinal lesion is rather superfluous and a physical examination almost unnecessary. They contend that a careful psychiatric history and psychological study are all that is essential in caring for a patient with gastrointestinal symptoms. I have seen patients with palpable protruding abdominal tumors who had had shock treatments and pale emaciated patients with profuse bloody rectal discharges who had had repeated psychoanalyses. In all cases sedatives and tranquilizers are considered indispensable. They are being prescribed indiscriminately for any symptoms and often do harm.

It is a fact that almost all patients with chronic gastrointestinal complaints present psychological problems. Any person experiencing frequent bowel movements and rectal pains, any patient with recurrent attacks of peptic ulcer symptoms or recurrent allergic manifestations, any woman fearful of repeated biliary colics, will become apprehensive, often discouraged and depressed, occasionally actually psychotic. In nearly all such cases when the condition has been diagnosed, proper treatment prescribed and gastrointestinal symptoms alleviated, it is remarkable how quickly the psyche is improved. Repeatedly we hear wives or husbands of peptic ulcer and ulcerative colitis patients remark that the spouse who had always been irritable and neurotic had suddenly improved so that married life had for the first time in years become a pleasure.

Though psychological care is an important part of the art of practicing medicine, it does not need to be carried out in the complicated fashion recommended by the modern psychosomatist. When a patient has been subjected to a careful diagnostic study, when he has had a demonstration by means of charts and diagrams of exactly what ails him, and the reasons for each step in treatment have been carefully explained to him, he will realize that his doctor is worthy of confidence. Such confidence is a most important factor in successful treatment. The doctor's evident

interest optimism and encouragement with praise for a patient's cooperation when he shows improvement and the pointing out of reasons for failure to improve because directions were not adhered to are also important psychological aids to treatment in caring for any patient. The more intelligent patient today is critical of his doctor; knows that he is only guessing when he prescribes for him without having studied his case and soon leaves him for a physician who will not treat him until he knows all about him. On the other hand the less intelligent patient the believer in miracles wants medicine at once and goes from doctor to doctor being told he has a "nervous stomach" and collecting prescriptions for anticholinergics sedatives cathartics and other drugs. Such a patient finally becomes a neurotic or a psychotic problem. There follow amateurish attempts at psychiatric treatment followed by specialized care by psychiatrists including psychoanalysis shock treatments and the inevitable tranquilizers. Such patients who really have somatic disease usually remain physical and mental wrecks until subjected to adequate medical study and treatment.

Real *neuroses* or *psychoses* may often be accompanied by gastrointestinal symptoms. These symptoms may have been the original cause of the psychological condition as mentioned before or may be due to various other causes perhaps even organic conditions of the nervous system. The most common symptoms associated with these psychiatric conditions and not based on any organic gastrointestinal disease include anorexias such as anorexia nervosa bulimia swallowing difficulties such as globus hystericus spasms such as pylorospasm often with vomiting and motility disturbances such as diarrheas to mention a few. In such cases forced feedings which sometimes must be given by tube or by vein attention to fluid and electrolyte balance and treatment by a psychiatrist are often invaluable.

In *psychotics* there may be a fear of poisoning embarrassment over being observed while eating or defecating and a variety of other inhibitions such as are seen in schizoid states. In depressive states symptoms are often referred to the abdominal organs and such complaints as abdominal pain anorexia or perverted appetite bitter taste and loss of weight with general weakness may be so outstanding as to mask the recurrent usually hereditary attacks of depression insomnia and a feeling of unreality which would clinch the diagnosis. At times during an attack of depression symptoms of an organic disease from which a patient suffered years before may come to the fore with no evidence of that disease now demonstrable. In all psychotics diets must be carefully supervised in order to avoid the results of overfeeding or actual starvation.

In *neuroses* a careful history of the original cause is often revealing. Feelings of anxiety fear guilt frustration insecurity inferiority and

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were due to *psychic stimuli* but in more recent years actual somatic diseases such as peptic ulcer and ulcerative colitis have been taken over by the psychiatrists with the contention that they are definitely the result of previous or present psychic trauma. The interesting scientific studies of Selye have demonstrated that stress whether psychological (emotional) or physical (trauma including surgical operations, infections and their sequelae) has a definite effect on the endocrine system. Instead of accepting this action as evidence of a physical (glandular) cause for the phenomena observed, instead of stimulating a search for possible hidden physical stresses such as chronic infections or other diseases, psychic stress alone has been emphasized and Selye's observations have been considered a confirmation of the theory of "mind over matter." This has hastened the development of the elaborate system of psychosomatic medicine with which we have to contend today.

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of the external surface of the organism which in its unicellular state obtained all its nourishment by absorption from the surface. Parasites occupying the lumen of the alimentary canal are said to infest it and the condition is called an infestation. When the parasites leave the lumen either traveling through the mucosa and thence to various parts of the body or invading such orifices as the trachea, the common duct or the pancreatic duct some authorities speak of "infection" but the term "infestation" is still applicable. Severe infestation with *Intamoeba histolytica* is also at times spoken of as an "infection." In many cases multiple infestations are encountered.

The *etiology* is variable. In some cases the infestation may be spread by direct ingestion of the parasite or its ova from human excrement; in others an intermediate host is necessary. In general, poor sanitation is an important factor and poor nutrition contributes to the susceptibility of the individual and his inability to cope with the invader.

The *symptoms* vary; some patients having no discomfort at all from the presence of the parasite, others appearing extremely toxic, undernourished and anemic and suffering pain with symptoms referable to the gastrointestinal tract or other systems which have been invaded. A variety of allergic symptoms may be caused by sensitivity to the parasite or its excretions.

*Physical examination* does not as a rule disclose anything which would establish a diagnosis of parasitism, but it should be carefully conducted in order to evaluate the patient's general condition and his ability to survive the treatment necessary to eliminate the parasite from the body. It is particularly important to have an accurate evaluation of the state of the cardiovascular system, the gastrointestinal tract and liver, urinary tract and blood. A patient with a history of allergies must be handled with great care.

*Diagnosis* of the specific parasitic organism or organisms depends usually upon careful, repeated stool examinations. Fresh specimens obtained by proctoscopy are particularly valuable. Parasites, their ova or cysts may be found and in the case of amebas, cultures can be grown. In some cases agglutination and complement fixation tests are reliable; in some, skin tests may be helpful. It must always be borne in mind that multiple infestations may occur and that therefore the finding of one parasite is not the signal for stopping the study. Search for parasites invading other organs will be discussed under the discussion of each parasite.

#### TREATMENT

Before attempting to destroy or eliminate a parasite, it is important to treat the patient. Any abnormal conditions found on careful study should be treated and the patient's general state of nutrition brought up

worries regarding marital infidelity sexual difficulties promiscuity or perversions conditions of employment relations to employer and fellow employees infractions of religious laws and many other sources of psychic stress may be discovered by means of a careful history and efforts must be made to correct them Today too much emphasis is being placed on prenatal infantile and childhood stresses as causes of disturbances in later life Such simple usual and natural procedures as weaning spanking extractions of teeth removal of tonsils and treatment of childhood injuries are blamed for diseases in later life A history of such mild long forgotten stresses can be obtained from any normal person

In any patient with gastrointestinal symptoms a complete study should include not only a thorough study of the gastrointestinal tract to determine whether organic disease is present and a search for evidences of endocrine imbalance but also a careful study to rule out symptoms of a real neurosis or a psychosis If either is found to be definitely present it should not be neglected The organic condition should be treated adequately by the clinician even though a psychiatrist has been engaged to follow the psychological aspects of the case Never should the treatment of any organic disease be entrusted to the exclusive care of a psychiatrist

## Intestinal Parasites

### General Discussion

In tropical and semitropical climates parasites are commonly found in the human intestinal tract In temperate and colder climates the more primitive protozoan organisms are much more rarely found and then in persons who have been in the tropics or have received their infestation originally from persons who were there The larger metazoan parasites are more generally distributed although many are endemic in certain areas

In general these parasites are animal organisms and therefore when confined to the intestinal tract can be considered to be living in symbiosis with the host They may simply share the food ingested and digested by the host When the host is taking a plentiful amount of food there is usually enough to supply the needs of the parasite without interfering with the nutritional status of the host Technically any material contained within the lumen of the alimentary canal is really outside the body the mucosa of the tube being merely an extension and duplication

## EPIDEMIOLOGY

Transmission occurs usually by contamination of food or drink by either the parasites themselves or their cysts or spores. They may thus be transferred from human or animal hosts. The method of transmission in amebiasis is shown in the chart (Fig. 11). Recognition of the parasites or their cysts in intestinal content, stools or scrapings from ulcers or crypts harboring them is usually difficult and requires careful and repeated study by an expert in parasitology. The stools to be examined may be first a casual specimen, then a warm specimen preferably passed in the laboratory, and then several specimens obtained after saline purgation or saline enema. The stools are examined by means of a fresh smear, stained smears, by concentration or flotation methods. In some cases cultures can be grown. The exact method of study should be left to the

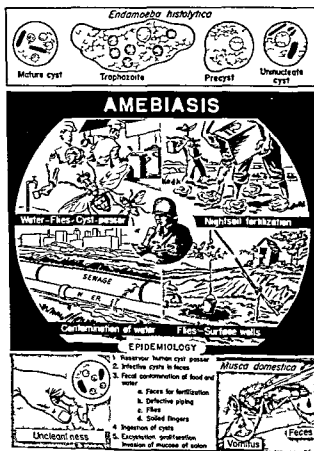


Figure 11 Epidemiology of amebiasis (Mackie Hunter and Worth Manual of Tropical Medicine 2nd ed.)

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to as nearly normal as possible so as to minimize the danger of the treatment

*Any poison used to destroy the animal parasite is also a poison to the host* With the host in poor condition the poison may cause death Research is going on constantly in an effort to find drugs which will get rid of the parasite efficiently without exposing the host to too great a danger of poisoning No ideal drug has yet been found No drugs are absolutely specific for any particular parasite and new ones are being tried out constantly Drugs used to destroy the parasite are called *parasitocides* Those destroying amebias are called *amebicides* worms *termicides* Drugs used to anesthetize a worm so that it may be eliminated by catharsis are called *vermifuges*

*Preparation of the gastrointestinal tract for the administration of the drug is important to assure a good result* Though a few parasitocides are efficient regardless of the quantity of intestinal content as in the case of amebicides in most cases of larger parasites a good result can best be accomplished when the drug can attack the parasite directly with the bowel empty or nearly empty A nutritious low residue diet such as our ulcer diet (p 267) for three days before treatment with a daily morning saline laxative will not as a rule deplete the patient too much and will provide the desirable conditions The drug should then be given on the morning of the fourth day after an all night fast It can be given by mouth or by duodenal tube Within a reasonable time after ingestion a saline cathartic should be given to wash out the dead or sick parasites All stools should be examined to find what has been expelled No food should be given until the cathartic has taken effect In some cases parenteral injection of a parasiticide can be given

### PROGNOSIS

There is no 100 per cent cure Some parasites are more easily eliminated than others but it is exceptional for no recurrences to be noted Even when the head of a tapeworm is found there may be another tapeworm surviving In all cases careful search for parasites and ova must be conducted at intervals for a year or more so that retreatment can be instituted before too great an infestation has been allowed to develop

### Protozoa

Several varieties of protozoa single celled animals are found in the human intestine both large and small Only a few are definitely pathological the others occurring without symptoms or with mild symptoms Many occur in connection with other parasites so that sometimes three or four different ones are found in the same patient

may ulcerate causing bloody diarrhea. The diagnosis is made by smears and cultures of blood and bone marrow, splenic puncture and various skin and complement fixation tests. The treatment consists in general cure and the use of antimonials and stilbamidine.

The ciliated *Balantidium coli*, rarely seen in this country, is usually harmless. It may live quietly in the lumen of the colon and cause no symptoms. It is transmitted as in the ameba by formation of cysts. Rarely it may invade the colonic mucosa, producing lesions resembling amebic ulcers and causing symptoms like those of amebiasis, even to severe dysentery and death. The diagnosis is made by stool examinations and scrapings from ulcers seen through the proctoscope. The treatment is the same as for amebiasis.

Amebae of five species have been found in the human intestine. It has always been considered that four of them, *Entamoeba coli*, *Endolimax nana*, *Iodamoeba buetschli* and *Dientamoeba fragilis*, cause no symptoms and do no harm, occurring mainly in regions where other protozoa are found. Recently *Dientamoeba fragilis* has been shown to infest the appendix causing appendicitis, especially in children. The principal importance of all these amebae lies in the necessity of differentiating them from the important pathogen, *Entamoeba histolytica*. The incidence of amebic infestation and its intensity vary in different parts of the world. Like other parasites, *E. histolytica* is encountered more in rural districts with poor sanitation, more in hot climates and in patients whose under-nutritious dietary habits result in poor general health. In this country, various estimates of its incidence have been made, varying from 2 to 40 per cent and more of the whole population. Conservative estimates consider the incidence in northern urban United States as between 1 and 4 per cent and in our Southern, more rural districts, as from 6 to 23 per cent, the latter in Alabama.

#### AMEBIASIS

Amebiasis is the term used to indicate an infestation with *Entamoeba histolytica*. It includes not only acute amebic colitis or dysentery, but also the milder, chronic form, as well as infestation of other organs to which the parasite has migrated. These amebae may live in peaceful symbiosis with the host for years. They usually locate in a quiet part of the bowel, like the sigmoid or cecum, hiding in small crypts produced by digestion of small areas of mucosa by their own enzymes. Feeding on red blood cells and bacteria, the active, motile amebae, known as trophozoites, become encysted when mature and the cysts are passed in the feces. These cysts can live for a comparatively long time outside of the body and when ingested by other human beings will become trophozoites and establish themselves. In the quiescent stage, with mild infestation, only the cysts will be expelled, usually in otherwise normal



laboratory. It is only by such repeated studies that their presence can be ruled out.

#### PROPHYLAXIS

Prophylaxis is important. Sterilization of stools of infested patients and thorough cleanliness of patient and attendants are imperative. Avoidance of uncooked foods and drinks in regions where protozoa are prevalent is necessary. All food handlers should be regularly examined with careful and repeated stool examinations insisted upon. Vegetable gardens should not be fertilized with human feces. Flies and other insects must be eliminated and sewage disposal must be thorough and frequently inspected. Treatments will be discussed for each parasite. Most of the innocuous ones are disposed of by antamebic therapy.

#### TYPES OF PROTOZOA

The harmless protozoa, those producing no appreciable gastrointestinal symptoms, include the flagellates of the trichomonas group. Five or six different varieties have been found at times. All show one or more long flagellæ making them motile. The sporozoa, spore forming and less motile, are seen in some Mediterranean countries. Included among this group are the malarial parasites, which do not inhabit the intestine.

The flagellate *Giardia lamblia* inhabits the small intestine, mostly in children and in the elderly. It is the most common flagellate occurring in the intestines and is frequently found with other parasites. There is still considerable difference of opinion about its relation to disease. Only one half of patients harboring this parasite have any symptoms. If large numbers of parasites are moving about the small intestine, it is conceivable that they may cause a feeling of peristaltic unrest, general nervous irritability, retrostaltic gastric symptoms and increased intestinal motility with diarrhea. The parasites have been found in duodenal contents and in biliary drainage material, so that it has been asserted that the biliary ducts may become occluded and cholecystitis and cholelithiasis may supervene. A giardial dysentery has also been described. The diagnosis is made from the finding of the parasites in the stools. The treatment, which has been almost 100 per cent successful in eliminating these parasites, consists in giving a course of Atabrine, as in the treatment of malaria,  $1\frac{1}{2}$  grains three times daily for five days, repeated once or twice at weekly intervals. They are also eliminated by the use of the drugs used for amebiasis.

*Leishmania donovani*, a protozoal organism, is the cause of kala azar, which is endemic in low lying tropical regions in various parts of the world. The organisms, also called Leishman-Donovan bodies, are found mainly in the reticuloendothelial system. The spleen becomes greatly enlarged, the liver less frequently. The small and large intestinal mucosae

larged and tender liver which is a common finding in dysentery even though there is no direct evidence of invasion. These findings disappear when the amebae are destroyed in the intestine. Occasionally however an amebic hepatitis can be recognized and an amebic abscess may form producing more or less severe symptoms. The hepatitis may be due only to an allergic reaction.

Other complications are rare. A liver abscess may rupture into the chest producing pleuritis or lung abscess. Brain abscesses may be secondary to these. Other rare complications include pelvic prostatic or rectovaginal fistulas, thrombosis of the vena cava or renal vein, pyelephlebitis, abscesses of the spleen, kidney, ovary, testicle and even in the skin. Severe hemorrhages are rarely encountered. Secondary bacterial infection is not uncommon. The complications vary in different climates and in accordance with the patient's general condition.

Complications may easily be misinterpreted. Intestinal obstruction due to cicatricial contraction resembles that due to other causes. Hepatitis may not be suspected as being due to amebiasis. Invasion of the chest may be mistaken for pneumonia, pleurisy or empyema. The other rare complications will produce no symptoms characteristic of amebic infestation except that a history of previous dysentery should occasion a suspicion. A history of living in the tropics or associating with persons from areas known to be infested is important.

### EXAMINATION

The patient may show no abnormal finding until the disease has been present for a long time. On the other hand as the patients are most frequently in a debilitated state before becoming infested, evidences of food and vitamin deficiencies may be found, neglected teeth, chronic upper respiratory tract disease and pulmonary, cardiovascular, renal and gastrointestinal lesions should be carefully searched for. Abdominal examination may show slight tenderness in both lower quadrants or along the whole colon. The liver may be more or less enlarged and tender and jaundice may occur. If an abscess has formed it may be palpable and if so may fluctuate or it may merely push the liver down. If it has ruptured into the chest the findings may be those of pleuritic effusion or pneumonia. Colonic amebomas may be palpable as small or large firm perhaps slightly tender masses and can be confused with cancer. An old thickened colon may be palpable. Obstruction and perforation produce the same picture as when due to other causes.

### Rectal Examination

Rectal examination may show spasm with digital examination. fistulas may be seen with a speculum. *Proctoscopy* in severe cases may rarely show moderate redness and edema due to proctosigmoiditis. The char-

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**feces** When large numbers of amebae have produced large and deep ulcers with undermined edges the bowel becomes irritated diarrhea is produced and the disturbed living trophozoites are expelled usually enclosed in small clumps of bloody mucus. The amebae are then sick and produce few cysts. They live but a short time outside of the body even when kept warm and moist.

### SYMPTOMS

As has been mentioned when the amebae are living in peaceful symbiosis with the host there may be no symptoms sometimes for years the host acting as a carrier by passing cysts in his feces. In some cases the host may have attacks of more or less severe symptoms usually in warm weather. Some activity in the colonies of amebae may cause irritation so as to produce retrostaltic symptoms such as anorexia heart burn belching nausea and vomiting often with constipation and with occasional bouts of diarrhea. Such patients with insomnia headache backache general aches and sweating easy fatigability discomfort in the lower abdominal quadrants and general toxic appearance are usually treated as psychosomatic cases. Others may be treated for peptic ulcer gastritis gallbladder disease appendicitis or merely "virus infection." Many cases are suspected of allergy or other food poisoning. With increasing activity of the amebae and the resulting greater irritation diarrhea may become constant with expulsion first of much mucus. There may at first be pain in the region of the cecum and sigmoid later severe cramps almost constant tenesmus and twenty to thirty discharges of bloody mucus with more or less soft or watery feces the picture of acute dysentery. In severe cases the acute dysentery is severe from the start after an incubation period of two weeks to three months. In either case the patient becomes markedly depleted dehydrated and anemic has more or less fever and may with difficulty be persuaded to eat or drink.

### COMPLICATIONS

Complications may occur as a result of prolonged or repeated active periods. In some cases the colon responds to the irritation by the formation of granulomatous masses called amebomas usually located in the cecum or sigmoid. These may greatly resemble cancer. In other cases the repeated ulcerations invading the deeper layers may produce perforations with peritonitis. Induration may occur with marked thickening of the colonic wall so that it may become as stiff as a garden hose. Cicatricial contractions may produce obstructions.

Inhabiting the colonic mucosa as they do some active amebae must frequently find their way into the mesenteric venules and be carried through the portal vein to the liver. Usually they probably fail to survive but their presence appears to be the cause of the moderately en-

lesions. Liver function tests usually show no abnormal findings even with considerable amebic hepatitis.

### SPECIFIC DIAGNOSIS

Examination of feces or scrapings from ulcers is the most important step in diagnosis. When ulcers are within reach of the proctoscope, as they are in most cases, the scraping is most satisfactory, although it may need to be done daily for several days before a positive smear is obtained. When daily proctoscopy is impracticable, the stools will have to be examined.

#### Stool Examination

In an *acute dysentery* the fresh, warm stool is absolutely necessary. It will not keep and must be taken to a nearby laboratory immediately or preferably passed into a container in the laboratory. A small fleck of bloody mucus or the fresh smear obtained by proctoscopy, unstained or possibly faintly stained with a little Lugol's solution, must be placed on a warm slide with a warm coverglass and preferably examined on a warm stage. I prefer not to add warm saline solution. Under low power, study of small streams of moving debris and blood cells will show the trophozoites with their actively progressive and directional motility, hyaline-like pseudopods being pushed out and the content of the whole cell pouring into them and with red blood inclusions. They are carried along in the stream, turning over and over, but fighting against being thus carried by clinging to its margins. They soon become sluggish in their movements, assume a globular form and die. The trophozoites are from three to five times the diameter of the red blood cells. Under high power the amebae can be seen better, but they move rapidly out of the field. If all three of these criteria for identifying trophozoites are applied, they will not be confused with macrophages. The trophozoites can usually be demonstrated in the first scraping from an ulcer. It may require repeated search in warm stools for a week or more before one can be sure that none have been missed by this method.

In the *chronic or asymptomatic stage*, trophozoites are but rarely seen, although a saline purge may at times bring down a small number. In these cases the cysts are being expelled, and their recognition will make the diagnosis. Recognition is a job for an expert in parasitology. Various methods of preparation are necessary. In specimens of stools, cysts can easily be seen, but it is necessary to differentiate the cysts of the various amebae. The cysts of *Entamoeba histolytica* can usually be identified. They form large and small cysts. Their size varies from one half the diameter of the red blood cell to two or three times its diameter or larger. Mature cysts are spherical, have a refractive wall and contain four nuclei. The cysts of *Entamoeba coli* are two or three times larger and

characteristic finding is the punched out ulcer with undermined edges and a base filled with necrotic material and mucus. These ulcers are usually from  $\frac{1}{8}$  to  $\frac{1}{2}$  inch in diameter in the early stages but may get larger or may merge with surrounding ulcers to form larger but still more or less discrete ulcers. In some cases there may be only a few widely separated ulcers seen on proctoscopy but their typical appearance and the comparatively mild reaction of the mucosa in general are quite characteristic. Scrapings made with a dull curet from the base and under the overhanging edges of these ulcers placed upon a warm slide and examined immediately will in a great number of cases establish the diagnosis by the finding of the motile amebae with enclosed red blood cells. Amebomas resembling polyps or cancer are rarely encountered and fistulas are unusual.

### Aspiration

In the case of suspected hepatic or pulmonary abscess aspiration can be safely performed but the amebae are not often found. They inhabit the walls of the abscess and even at operation may be seen only in material scraped from the wall. The fluid is usually viscid may be chocolate colored and may or may not contain blood. Liver biopsy is not usually advisable.

### X-ray Examination

X-ray studies of the gastrointestinal tract by means of a gastrointestinal series and barium enema may show no abnormal findings may demonstrate a colonic irritability and hypermotility or may show the characteristic hypertonicity and thickening of the wall and fuzzy appearance of the mucosa characteristic of ulcerative colitis from any cause. Rarely the ulcer craters can be seen. In the old cases thickening of the wall with shortening of the colon the so called garden hose appearance or areas of deformity and kinking due to cicatrices are easily demonstrable. Amebomas produce defects resembling carcinoma which often disappear under treatment. In *hepatic amebiasis* scout films may show simply an enlarged liver or the protuberance of an abscess may be seen. If on the upper surface the abscess may cause elevation of the diaphragm and if it has ruptured into the chest signs of pleuritis effusion and pneumonitis may be evident in chest x-rays.

A complete x-ray study is of course of value in determining whether any lesions other than amebiasis may be coexistent.

### Laboratory Tests

These would include blood cell counts which will usually show an anemia and frequently a leukocytosis and increased sedimentation rate. Gastric analysis may be of value in discovering gastric or duodenal

was still widely used although its tendency to produce vomiting made it undesirable. Its alkaloid *emetine* was tried and was found to produce rapid and spectacular symptomatic relief even in cases with hepatic and other complications. The amebae however were found to thrive even after its prolonged administration. Later it was found that *emetine* is a definite poison to heart muscle and may cause death from heart failure. More recently it has been shown that daily doses of 1 grain of *emetine hydrochloride* intramuscularly for ten days rarely cause any appreciable heart damage although daily cardiac observation is recommended with immediate withdrawal in the presence of falling blood pressure, tachycardia or electrocardiographic changes. It is used today only for *extraintestinal amebiasis*. I prefer to use it if at all for not more than four to six days and combined with other amebicides.

*Other amebicides* include various chemical combinations of arsenic and iodine and quinine. Antibiotics have also been tried as have sulfonamides.

The *arsenic compounds* have included Carbarsone, Stovarsol and Salvarsan and while efficient have been largely abandoned because of the possibility of hepatic injury. A new preparation, Milibis bismuth glycolylarsanilate, has been used with considerable success. It is said to be insoluble and therefore nontoxic. It has been recommended in dosages of 0.5 gm tablets three times daily for seven days repeated after an interval of ten days. Chloroquine phosphate (Aralen) three tablets of 0.25 gm each for the seven days combined with the Milibis is supposed to be the most effective treatment. Aralen is given for its general effect on extraintestinal amebae. Quinacrine (Atabrine) is not as effective.

The *iodine compounds* have included chiniofon, vatren, vioform and more recently diodoquin. It has been questioned whether the iodine or the oxyquinoline contained in these preparations is the antiamebic factor. I have found chiniofon in doses of 1 gm or 16 grains (4 enteric coated tablets of 4 grains each) three times daily for eight days to be effective although at times it may irritate and cause a diarrhea. This however can usually be differentiated from the diarrhea caused by the parasite. It must be treated like diarrhea from other causes. Another eight-day course is given after an interval of ten days. In addition a daily retention enema of 300 cc of 1 per cent chiniofon in water is of value in intense rectosigmoid infestation. Diodoquin in dosage of one tablet of 650 mg three times daily for two courses of ten days each is equally effective with less tendency to cause diarrhea.

*Antibiotics* of various kinds have been exploited and later abandoned. The theory of their use was that with amebae feeding largely on intestinal bacteria the destruction of these bacteria would starve the amebae to death. It was also felt that some of the antibiotics might exert a direct

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contain eight nuclei those of the other amebæ look quite different. As cysts are usually not being passed regularly examination of daily stools for a week or ten days some fresh casual stools others after a saline laxative a saline enema or a specimen obtained from the sigmoid by suction through a proctoscope are usually necessary before a negative report can be made.

### Complement Fixation Tests

In the hands of some parasitologists complement fixation tests have been reported as 90 per cent efficient by others as of little value. Antigens are difficult to prepare. Such a test in reliable hands is of great value when the amebæ have migrated from the intestine as in hepatic amebiasis.

### Cultures

Cultures have been successfully prepared. The best results have been obtained by the combination of culture media and bacteria although growth has been obtained without the bacteria also. Here again there is a vast difference of opinion as to the value of cultures.

### Skin Tests

These have so far been of no definite value.

In *summary* amebiasis must be suspected in any patient with indefinite symptoms or actual disease of the gastrointestinal tract or in cases of diarrhea especially if bloody in hepatitis pleurisy and pneumonia especially if the patient has been overseas or in tropical countries or has worked or lived among people coming from countries known to be infested with parasites. Even though food allergy has been found to cause diarrheas and even though bacillary dysentery has been definitely diagnosed the possibility of accompanying amebiasis must always be considered. Finding of the *Entamoeba histolytica* is the only certain way of differentiating amebic colitis from that caused by schistosomal and balintidial dysentery. Proctoscopic diagnosis by scraping is most useful.

### TREATMENT

Treatment is necessary not only for amebiasis with a definite symptomatology but also for asymptomatic cases for carriers and even for prophylaxis in persons who have been exposed to possible infestation. As yet no real specific amebicides have been discovered although some drugs have been described as up to 90 per cent efficient in series of cases.

Before the ameba had been demonstrated as its cause dysentery was successfully treated with ipecac in India. For many years afterward it

was still widely used although its tendency to produce vomiting made it undesirable. Its alkaloid *emetine* was tried and was found to produce rapid and spectacular symptomatic relief even in cases with hepatic and other complications. The amebae however were found to thrive even after its prolonged administration. Later it was found that emetine is a definite poison to heart muscle and may cause death from heart failure. More recently it has been shown that daily doses of 1 gram of *emetine hydrochloride* intramuscularly for ten days rarely cause any appreciable heart damage although daily cardiac observation is recommended with immediate withdrawal in the presence of falling blood pressure, tachycardia or electrocardiographic changes. It is used today only for *extraintestinal amebiasis*. I prefer to use it if at all for not more than four to six days and combined with other amebicides.

*Other amebicides* include various chemical combinations of arsenic and iodine and quinine. Antibiotics have also been tried as have sulfonamides.

The *arsenic compounds* have included Carbarsone, Stovarsol and Salvarsan and while efficient have been largely abandoned because of the possibility of hepatic injury. A new preparation, Milibis, bismuth glycolylarsanilate, has been used with considerable success. It is said to be insoluble and therefore nontoxic. It has been recommended in dosages of 0.5 gm. tablets three times daily for seven days repeated after an interval of ten days. Chloroquine phosphate (Aralen) three tablets of 0.25 gm. each for the seven days combined with the Milibis is supposed to be the most effective treatment. Aralen is given for its general effect on extraintestinal amebae. Quinacrine (Atabrine) is not as effective.

The *iodine compounds* have included chiniofon, yatren, vioform and more recently diodoquin. It has been questioned whether the iodine or the oxyquinoline contained in these preparations is the antiamebic factor. I have found chiniofon in doses of 1 gm. or 16 grains (4 enteric coated tablets of 4 grains each) three times daily for eight days to be effective although at times it may irritate and cause a diarrhea. This however can usually be differentiated from the diarrhea caused by the parasite. It must be treated like diarrhea from other causes. Another eight-day course is given after an interval of ten days. In addition a daily retention enema of 300 cc. of 1 per cent chiniofon in water is of value in intense rectosigmoid infestation. Diodoquin in dosage of one tablet of 650 mg. three times daily for two courses of ten days each is equally effective with less tendency to cause diarrhea.

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effect upon the amebic. However recurrences have been more frequent following the use of antibiotics alone than after amebicides. They can be used in combination with amebicides when a secondary infection in the bowel or elsewhere is suspected. The tendency of broad spectrum antibiotics to cause lower bowel irritation with diarrhea and pruritus makes it wise to use them with caution. Sulfonamides are no longer used.

In *summary* it is best to use as directed above either chiniofon or diodoquin in two courses ten days apart or to try the newer combination of Milibis and Arlen. In some cases one course of each may be tried with an interval of ten days between them. Emetine is to be reserved for patients with severe hepatic symptoms and then preferably used for only four to six days or less combined with the other amebicides.

Abscesses in the liver, chest or elsewhere can be aspirated repeatedly until all the necrotic material has been removed and will eventually heal spontaneously if the patient is getting adequate amebicidal treatment. Washing out the abscess cavity with emetine was formerly recommended but is now considered unnecessary. Radical operations have never been satisfactory, often resulting in severe complications and possibly death.

For *perforation* or *obstruction* surgical intervention is definitely indicated. In severe colonic deformities resections may be required.

### General Treatment

Before and during the specific treatment for the amebiasis the patient's general condition must be kept at an optimum level. Any diseased conditions found in the complete study as recommended should be treated preferably even before amebicidal therapy. An adequate well balanced diet of the type recommended for liver disease is of value (p. 481). Vitamin and mineral supplements are important. The secondary anemia requires attention, ferrous sulphate being most helpful. Transfusions may be required. The need for prophylaxis which has already been discussed is most important.

### PROGNOSIS

Amebiasis is a serious condition. I once heard a famous parasitologist Dr. Patrick O'Conner say that the infestation is rarely thoroughly eradicated and that most patients would eventually die of it. He himself lived up to this tradition. Even today it is difficult to be sure of a cure in spite of repeated stool studies after cathartics. Patients considered cured may suddenly suffer a severe dysentery in summer or on a trip to the tropics. It is at times difficult to decide whether such a patient has been carrying the parasites or has ingested new ones. The prophylactic use of diodoquin while residing in infested districts is not invariably effective.

## Metazoa (Helminths)

Metazoa are multicellular parasites. The most common metazoa are the helminths or worms included in three groups: roundworms, tape worms and flukes. Some are endemic in certain parts of the world, some are universal in distribution. What has already been said about the factors tending to promote infestation of parasites in general applies also to these. In some cases dietary habits, especially the customary eating of uncooked foods, is an added factor.

## NEMATODES

Nematodes include several distinct parasites. The individual worms are recognized by the finding of their ova, which differ sufficiently to make a definite diagnosis. Figure 12 shows the characteristics of the ova of each type of worm now to be described in this section.

## ENTEROBIUS VERMICULARIS

*Enterobius* (formerly *Oxyuris*) *vermicularis*, also called pinworm, thread worm and seatworm, is universal in distribution. It has been estimated to infest from 30 to 50 per cent of children, 25 per cent of them of so-called best families. It has been said to get into the seats of the mighty. Infestation occurs from the swallowing of eggs, which mature in the small intestine, the adults remaining in the distal ileum and proximal colon for seven or eight weeks. Rarely they have been found in the appendix, causing inflammation there. The male dies after fertilizing the female, which lays eggs which pass down with the feces. At the end of her life the female migrates at night to the perianal region and deposits eggs in the skin folds, causing the severe itching which is the principal symptom of this infestation. It may be the cause of night mares and extreme nervousness, with anorexia and loss of weight and strength. The patient will always manage to scratch the area, even if tied down. The eggs, getting under the fingernails, are carried to the mouth, thus constantly renewing the infestation. The complete cycle takes two or three weeks. Deposited on food or drink, the eggs will infest others.

## Diagnosis

Ova are rarely found in the feces, but are recovered from the perianal region or from under the nails (Fig. 13). Live female worms may at times be seen on the surface of feces or crawling in the perianal region. They are white and about 1 cm. long. The best method of finding ova or worms is to prepare a swab by wrapping a piece of Scotch tape, sticky side out, around the tip of a wooden tongue depressor or the closed end of a test tube and pressing it into the stretched-out perianal crypts. The cellophane tape is then unwrapped, placed directly upon a

## 146 Diseases Affecting Entire Gastrointestinal Tract

effect upon the amebae. However, recurrences have been more frequent following the use of antibiotics alone than after amebicides. They can be used in combination with amebicides when a secondary infection in the bowel or elsewhere is suspected. The tendency of broad spectrum antibiotics to cause lower bowel irritation with diarrhea and pruritus and makes it wise to use them with caution. Sulfonamides are no longer used.

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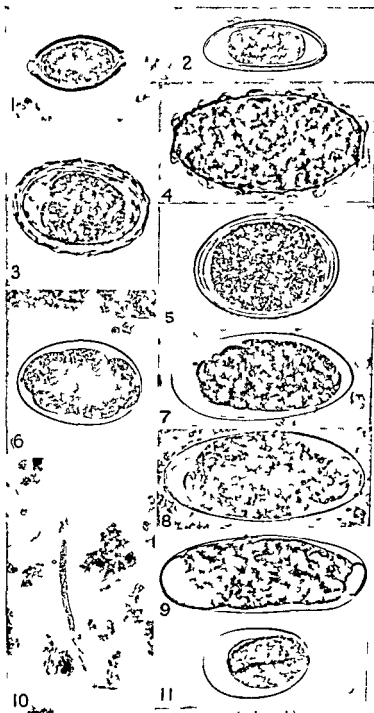


Figure 12 (See facing page for legend )

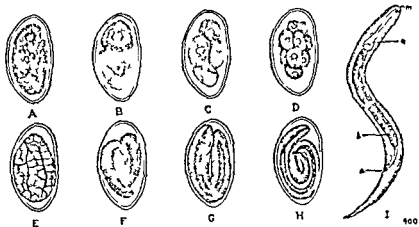


Figure 13 Stages in the development of the larva of *Enterobius vermicularis* (After Leuckart from Belding Textbook of Clinical Parasitology)

Lantern slide and examined under the microscope. The characteristic finding of ova showing contained embryo worms may often be obtained on a first examination although repeated examinations may be necessary. This type technique is also used after treatment to determine the effect. At least ten such examinations with negative results at two or three day intervals starting ten days or two weeks after treatment are considered necessary to pronounce a patient cured. The mother or other attendant can be taught to use the swabs and bring them to the doctor or laboratory.

### Treatment

Various methods of treatment have been used over the years anthelmintics having been used both orally and rectally. Failure of treatment is usually due to reinfestation from the patient himself or from infested playmates. The entire family and all contacts must be treated at the same time and efforts should be made to prevent scratching and soiling of fingers at defecation. General measures for preventing infestations and reinfestations have been described before. Frequent swab examina-

FIGURE 12 Some common nematode eggs (1) whipworm *Trichuris trichiura* (2) pinworm *Enterobius vermicularis* (3) large roundworm *Ascaris lumbricoides* fertilized egg (4) *Ascaris* infertilized egg (5) *Ascaris* decorticated egg (6) hookworm egg (7) immature egg of *Trichostrongylus orientalis* (8) embryonated egg of *T. orientalis* (9) egg of *Heterodera marioni* a plant nematode which sometimes is found in soil (10) habit form larva of *Strongyloides stercoralis* the stage usually found in stool (11) egg of *S. stercoralis* rarely seen in the stool. All figures 500  $\times$  except (10). Nos. 5 and 16 courtesy of the Photographic Laboratory AMSCS photos by Milt Chalk. Nos. 7, 8 and 9 courtesy of Dr. T. B. Lagish Mayo Clinic. All others courtesy of Dr. R. L. Roudabush, Ward's Natural Science Establishment, Rochester, N.Y. photos by T. Romanik.



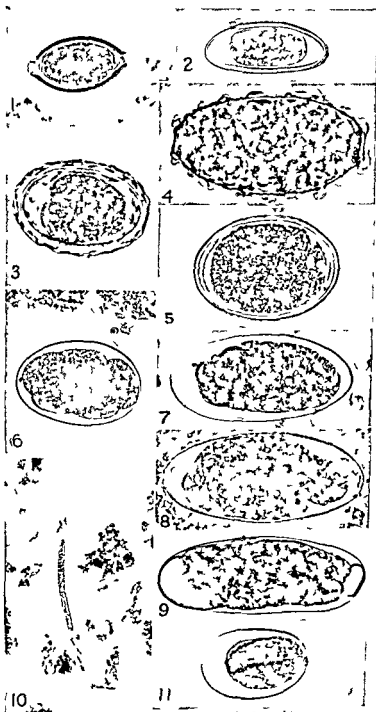


Figure 12 (See facing page for legend)

swallowed they settle in the duodenum in the same way as hookworms. Some larvae which have been passed may develop into free living adults and produce free living colonies although eventually infective stage larvae are again produced. In some cases the host may become reinfested by his own larvae penetrating the intestinal wall or perianal skin and going through the same stages as those entering the skin from outside. This is called autoinfection. Man is the only host.

When the larvae enter the skin usually of the foot there is a pricking sensation followed by itching and redness. A week later the invasion of the lung causes a cough occasionally pneumonia. A month or so later as larvae are being expelled there may be epigastric pain and diarrhea with mucus and rarely blood sometimes alternating with constipation. Occasionally the worms may invade the circulation with parasitemia and death. With infestation prolonged by autoinfection the patient may become toxic and depressed. A leukocytosis with marked eosinophilia up to 75 per cent should occasion a suspicion of this condition although in late stages both may be greatly reduced.

Diagnosis is made by the finding of ova or larvae which must be differentiated from those of the hookworm. They are usually found in the feces rarely in the sputum.

### Treatment

Gentian violet (medicinal) is the usual drug used but is not a specific. One half grain (0.006 gm) enteric coated capsules are available. Two capsules are given three times daily before meals until 50 grains (15 gm) have been taken. One or two such courses at intervals of ten days will expel worms in the majority of cases. If this has not been accomplished or if the patient cannot take the capsules without pain and vomiting the drug can be given through a duodenal tube. On a morning in a fasting state 25 cc of 1 per cent solution are slowly instilled into the duodenum. In three hours a transduodenal lavage of 500 cc of a solution of sodium phosphate and sodium sulfate 5 per cent of each can be given warm and very slowly to produce rapid and effective purgation and removal of dead or poisoned worms as well as the irritating drug (p. 49).

Prophylaxis consists in proper disposal of infested feces and avoiding barefoot walking in endemic areas.

### ASCARIS LUMBRICOIDES

*Ascaris lumbricoides* commonly called the round worm and causing ascariasis is our most common parasite and occurs chiefly in children. Like other parasites it is found mostly in warm climates but also in the north where with unsanitary conditions feces can contaminate food and water. The female is 8 to 10 cm long looks like an earthworm but

tions must be kept up after treatment to make sure about cure or reinfestation

*Drugs* Various drugs have been tried and found wanting *Gentian violet* in the form of enteric coated tablets and jellies or suppositories of rectal use is spectacular in its staining effects but falls far short of being a cure *Hexylresorcinol* has been used with considerable success It is given orally preferably in hard gelatin capsules (Crystoids) These capsules contain 0.2 gm each and 1 gm (5 capsules) is the dose for adults slightly smaller for children A saline purge is used the afternoon before and three hours after the drug has been taken A supplemental retention enema of 300 cc of 1:1000 solution (full strength ST 37) retained for twenty minutes has also been used Two or three such courses may be given at weekly intervals

*Piperazine citrate* in 2 gm doses daily before breakfast for seven days has been found effective in 97 per cent of cases

*Promethazine hydrochloride* an antihistaminic marketed under the name of Phenergan has recently been found to be particularly effective It is administered at bedtime without previous fasting in a single dose of 125 mg (10 tablets of 12.5 mg each) For small children the tablets can be crushed and mixed with jelly or flavored syrup The drug is non-toxic having been used in some mental disorders in doses up to 1.5 gm a day for long periods without any adverse effects Rarely in small children nightmares may occur after administration of the drug but as they are also frequent symptoms of the infestation itself can be discounted No drowsiness has been noted the next morning In well over 90 per cent of cases a single treatment cleared up the infestation Being harmless the drug can be given to all members of a family or group to prevent reinfestation

An old rectal treatment which disposes of the worms in the colon effectively consists in instilling 100 cc of a 1:1000 solution of *corrosive sublimate* leaving it in situ for exactly ten minutes and then washing out the colon thoroughly with saline enemata Repeating this procedure the next day will usually assure an excellent result although it is of course necessary to use all precautions against reinfestation

#### STRONGYLOIDES STERCORALIS

*Strongyloides stercoralis* which causes strongyloidiasis are tiny round worms found in warm climates but also occasionally as far north as New York The females about 2.2 mm in length inhabit the villi and glands of the mucosa of the human duodenum and jejunum where eggs are laid and where they are hatched The larvae burrow out to the intestinal lumen and are passed in the feces They grow in the ground and become filariform and infective entering the human skin traveling to the right side of the heart and lungs bronchi and trachea Then being

a gastrointestinal x ray study done for the abdominal symptoms may reveal the worms either as tubular negative shadows in the barium filled small intestine (Fig. 14) or as positive shadows with barium in the intestinal tract of the worm showing after the intestine has emptied itself. An eosinophilia is always suggestive of a parasitic infestation.

### Treatment

*Prophylaxis* has been described in the general section on parasites. It also may be postulated that during the migratory stage possibility of infestation may occur with kissing.

Various anthelmintic drugs have been tried in the past and none found to give really good results. Of these hexylresorcinol in the form of Crystoids 1 gm (5 capsules) followed in three hours by a saline purge and repeated in two days has been reported as giving 85 per cent of cures. More recently piperazine salts in a single mid-afternoon dose of 3.5 gm in tablets or chewable wafers have produced 86 per cent of cures and when given in 2 gm doses daily for two weeks 100 per cent of cures.

For some of the serious complications operation may be necessary. Operations have been performed for intestinal obstruction of unknown origin and worms found to be the cause.

### Prognosis

In uncomplicated infestations the results of treatment are usually good although a second course of treatment may be necessary. Even without treatment the worms will die out in a year or so and the patient will get well unless reinfestation takes place.



FIGURE 14 Roundworms (*Ascaris lumbricoides*) X ray films of small intestine. a Arrows point to negative shadows of the worms in the barium. b Arrows show barium in lumen of worms after barium meal has passed. (v) appendix.

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is hard and wiry to the touch. It may be cream colored, pale pink or light brown. The male, which is smaller, has a curved tail with a spicule at its tip; the larger female has a straight tail. They live in the small intestine. In rare cases only one is present and there may be dozens. The female lays large numbers of eggs, up to one quarter million daily, some fertile, some infertile. The fertile eggs have a tough covering and have been known to remain viable for three or four years (Fig. 12). They infest only the human organism. They develop embryos while still outside the body and when this has occurred are capable of growing in man when ingested. Human feces infected with these embryonated eggs mixed with soil in which children play is the common source of infestation. When ingested, the eggs hatch in the small intestine and the larvae penetrate the intestinal wall and enter venules or lymphatics. They are carried to the right side of the heart thence to the lungs, out through alveoli, bronchi, trachea and larynx across the epiglottis into the esophagus and through the stomach to settle finally in the small intestine, the entire process taking ten days. In ten weeks they become adult worms and are ready to start the whole cycle again. When larvae in large numbers pass through the lungs they may produce *ascaris* pneumonia, often difficult to diagnose but usually clearing up rapidly.

### Symptoms

With only one or two worms in the intestine there may be no symptoms but more worms will cause annoying creeping feelings with occasional acute colicky pains. Their bulk may produce anorexia and retrostaltic symptoms. Large numbers have been known to cause actual intestinal obstruction. They have been known to crawl into the stomach and be vomited; they have traveled up the esophagus and into the respiratory system, spinal cord and brain. They have been found in the biliary tract, pancreas, liver, appendix and fallopian tubes. They may cause mucosal ulceration with bleeding or may penetrate to the peritoneum. At times adult worms may be passed spontaneously or may protrude from the anus so that with a firm grasp they may be pulled out. Even in the absence of symptoms from complications the worms cause a more or less marked toxemia, often with mild fever, secondary anemia and eosinophilia, urticaria, eyelid edema and nervous and mental symptoms.

### Diagnosis

When worms have been passed by rectum or vomited the diagnosis is easy. Otherwise the abdominal symptoms, the cachectic appearance and mental symptoms may result in all kinds of diagnoses. In children worms must be suspected with any mysterious symptoms. Repeated stool examinations by an expert may disclose the typical ova, although if only one or two male worms are present no ova will be found. At times

a gastrointestinal x ray study done for the abdominal symptoms may reveal the worms either as tubular negative shadows in the barium filled small intestine (Fig 14) or as positive shadows with barium in the intestinal tract of the worm showing after the intestine has emptied itself. An eosinophilia is always suggestive of a parasitic infestation.

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FIGURE 14. *Roundworms (Ascaris lumbricoides)*. X-ray films of small intestine. a. Arrow point to negative shadows of the worms in the barium. b. Arrows show barium in lumen of worms after barium meal has passed. (A) appendix.

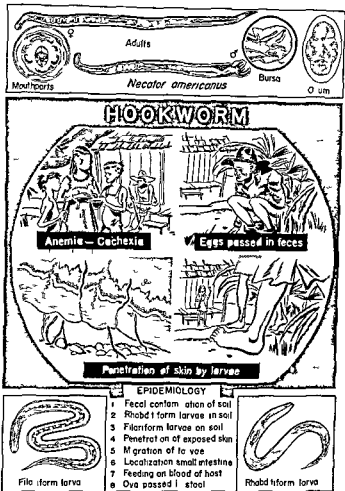


Figure 15 Epidemiology of hookworm disease (Mackie Hunter and Worth Manual of Tropical Medicine 2nd ed.)

#### NECATOR AMERICANUS (ANCYLOSTOMA DUODENALE)

Hookworm infestation called *uncinariasis* may be caused by the Old World *Ancylostoma duodenale* or the New World *Necator americanus*. *Ancylostoma braziliense*, a parasite of dogs and cats, also affects man in Brazil. In this country it occurs mostly in the south in regions where people habitually go barefoot. The worms are small, barely visible to the naked eye, being only 8 or 10 mm in length. They inhabit the small intestine, mainly the jejunum, attached to the mucosa and sucking the blood of the host. Individual worms may live up to five or six years. Impregnated eggs pass out through the feces daily by the thousands and if deposited in warm, moist soil will develop into wriggling larvae. These penetrate the skin when the foot or hand is pressed into the soil.

enter the venules travel to the right side of the heart lungs and trachea. Some are coughed up others are swallowed and return to the small intestine where they grow into adult worms. The whole process from egg to egg takes about fifty days (Fig. 15).

### Pathology

The skin at the site of the larval penetration develops vesicles then papules and with infection pustules a condition called "ground itch." It is seen mainly in children farmers gardeners and miners as would be expected. On reaching the bronchial tree it may cause bronchitis or even pneumonitis. The blood sucking produces a secondary anemia as does ulceration due to secondary infection when the worms abandon their sites.

### Symptoms

The skin symptoms appear first followed by the pulmonary symptoms. A cough may be overlooked. Gastrointestinal symptoms may be mild of the retrostiltic type or with ulceration may produce hunger pains and colicky feelings. A perversion of appetite—the desire to ingest earth chalk or wood—may occur. The patient loses weight and strength and becomes apathetic dizzy sometimes almost comatose. The hair becomes coarse and falls out the skin and mucous membranes become pale and often edematous. Sexual desire and menstruation may cease and abortion may occur. The eyes may resemble those of a fish.

### Diagnosis

In regions where the parasites are endemic and in patients known to go barefoot or to work in moist soil a marked anemia and weakness should engender a suspicion of *uncinariasis*. The hypochromic microcytic anemia is often extreme with hemoglobin readings as low as 10 or 15 per cent and red blood cells less than one million. Occasionally fractional gastric analysis will show in achlorhydria and blood may be found in regurgitated duodenal contents. The stools which always contain occult blood should be carefully studied by an expert for the presence of characteristic ova (Fig. 15). Occasionally especially after a cathartic these tiny worms may be seen wriggling in the feces.

### Prophylaxis

Prophylaxis is important. In addition to the precautions described before thorough treatment of infested persons and the wearing of shoes and gloves in endemic areas is important.

### Treatment

This is one disease in which it is imperative to prepare the patient for the use of the anthelmintic or he may die during the treatment. In



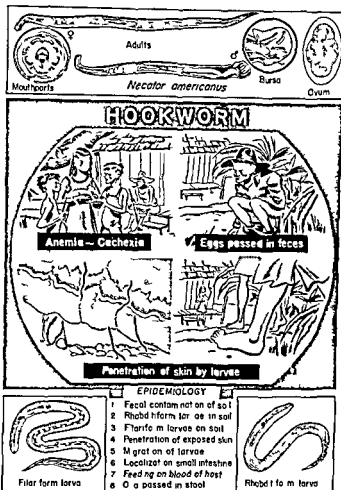


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### Treatment

No specific cure has been found. Patients should be treated with anthelmintics suggested for other nematodes.

### TRICHINELLA SPIRALIS

*Trichinella spiralis* which causes trichinosis belongs in the same group but stays in the intestinal tract only a short time. It is caused by ingestion of raw or only partially cooked pork from pigs infested mostly by eating garbage infested with larvae. Modern treatment of such garbage preferably by boiling or the abolition of the custom of feeding any garbage to pigs is aimed at eradication of trichinosis entirely. The larvae mature in the human duodenum; the females are fertilized there while the males die and for six or seven weeks the females embedded in the mucosa pass large numbers of larvae. Many of these enter the blood stream, travel mostly to voluntary muscles where they become encysted and then calcify, meanwhile causing intense muscle pains.

In the first eight or ten days while the females are invading the mucosa causing duodenitis and jejunitis, symptoms resembling acute food poisoning are produced, occasionally accompanied by bleeding. At this stage larvae have been found in the stools. If larvae enter the heart muscle, lungs or brain, death may ensue.

Diagnosis is usually made by finding the encysted larvae in painful muscle, although repeated biopsies may be necessary to locate them. A high eosinophilia (up to 50 per cent) is suggestive. Skin tests are fairly reliable. Sensitization to the parasite may produce a train of allergic symptoms.

### Treatment

*Prophylaxis* is most important. General sanitary measures described for all parasitic infestations are necessary. The relation of garbage in the diet of pigs has been mentioned. Sterilization of pork by thorough cooking is the safest preventive. Prolonged quick freezing to 27° C (minus 16.6° F) for thirty-six hours has been shown to kill the larvae, but such prolonged quick freezing is not practicable. Exposure of pork to gamma radiation is also effective but impracticable.

There is no specific treatment for trichinosis. *Symptomatic treatment* of cardiac, respiratory or cranial symptoms and relief of pains in muscles is about all that can be done. Corticosteroids will afford relief. If in the acute stage larvae are recovered in the stools, an attempt should be made to destroy and eliminate as many as possible by transduodenal injection of hexylresorcinol, 100 cc of 1 per cent solution while fasting, followed by transduodenal lavage with 500 cc of hypertonic saline solution (5 per cent each of sodium phosphate and sodium sulfate) given warm and

severe cases an adequate normal balanced diet with added vitamins and iron should be given for a couple of weeks before giving the anthelmintic. Transfusions may be required. Various drugs have been used to get rid of the worms, most of them poisonous to the host. I have seen a patient killed with oil of chenopodium, another almost killed with carbon tetrachloride. The safest drug today is hexylresorcinol. It should be used as I have recommended in enterobius infestation, although only a cleansing saline enema is required afterward (p. 150). This drug is sufficiently innocuous to be used again if ova continue to be found in the feces. It is of value because it will also dispose of other worms which may be present at the same time.

### Prognosis

The results of careful treatment as outlined are usually excellent.

### TRICHURIS TRICHIURA

*Trichuris trichiura* or *Tricocephalus trichiurus* called the whipworm and causing trichuriasis is common in moist regions. Infestation is produced by the ingestion of eggs found in moist soil, in water or on garden vegetables. The worms are from 30 to 50 mm. long and live in the cecum, appendix and ascending colon, imbedding themselves partly in the mucosa. The females lay eggs by the thousand which expelled in the feces will mature in a month in moist soil, ready to be ingested and cause a new infestation. With a mild infestation there may be no symptoms, but in the presence of large numbers of worms abdominal discomfort, anorexia, nervous irritability and diarrhea have been known to occur. In the appendix they may cause appendicitis. Eosinophilia up to 15 or 20 per cent is a common finding. Stool examination by an expert will disclose the characteristic eggs (Fig. 121).

Hexylresorcinol is the most effective remedy. It is best given as described under *Enterobius vermicularis* (p. 150). Stools must be checked for ova at regular intervals.

### TRICHOSTRONGYLUS ORIENTALIS

*Trichostrongylus orientalis* rare in this country but endemic in Turkey and Asia is a nematode inhabiting the upper small intestine. Soldiers returning from the Orient have been found with this infestation. Infestation is produced by ingestion of the filariform larvae. It is a parasite of goats and sheep which expel the ova, contaminating soil and food. The ova are found in the feces and greatly resemble hookworm ova but are larger. Infestation causes an eosinophilia usually of 15 per cent but occasionally as high as 40 to 70 per cent.

Symptoms consist of abdominal and lower back pains, bouts of diarrhea, weakness, fatigue, emaciation and dizziness.

velop an inordinate appetite. There may be moderate anemia occasionally severe in fish tapeworm cases. Eosinophilia is not a constant finding. Adults often complain only of the embarrassment caused by expulsion of single proglottids or strings of them from the rectum which occurs at more or less irregular intervals. Somatic infestation which occurs mostly in fish tapeworm infestation will give symptoms in the organs involved.

Physical examination is negative except for the findings in somatic infestation which is fortunately rare.

### Diagnosis

When proglottids are passed spontaneously or after catharsis they can be recognized by pressing them between two glass slides and studying them with a hand lens. Differentiation among the three types of worm is made by noting the size and shape of the proglottids and counting the number of branches of the uterus seen in each segment (Fig. 16). The fish tapeworm has short broad segments with few branches; the pork tapeworm has longer segments with five to ten branches; and the beef tapeworm still longer segments with fifteen or twenty branches. The heads vary somewhat in size and appearance and can be differentiated

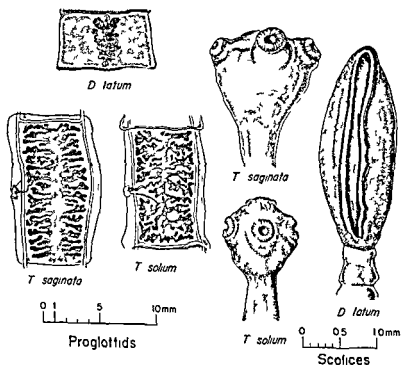


Figure 16 Scolices and gravid proglottids of some tapeworms of man (Mackie Hunter and Worth: Manual of Tropical Medicine, 2nd ed.)

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very slowly (p 49) Allergic symptoms are treated in the same way as allergy from other causes

### CESTODES

Cestodes or tapeworms are flat worms the adults of which are like ribbons or tapes Infestation is called teniasis The different species vary in length from 5 to 30 feet They are transmitted from animal hosts to man They occur almost universally The adult worm consists of a tiny head or scolex which is attached to the mucosa of the upper small intestine of man a neck in unsegmented portion from which develops a long row of segments or proglottids which get larger and more mature the farther they are from the neck All segments are hermaphroditic containing both male and female reproductive organs They may live for years if undisturbed The eggs expelled from the gravid segments in tremendous numbers are microscopic in size When eaten by a susceptible animal host or in the case of the fish worm two hosts first a crustacean then the fish they develop into larvae in the intestinal tract and migrate into the tissues of the host In the tissues they become encysted The cysts or cysticerci contain numerous scolices When the infested flesh of the host is eaten raw or underheated by man the cysts are digested and the scolices are freed These scolices or heads attaching themselves to the mucosa begin a new cycle which takes two or three months to complete The tapeworm usually causes little or no damage to the intestine living in peaceful coexistence with the host Rarely an autoinfestation may take place as a result of swallowing the eggs or regurgitating gravid proglottids into the stomach in which case serious damage may result from man's becoming also the intermediate host This condition is known as *somatic teniasis* The larvae may invade many organs including the liver

### Types of Tapeworms

There are three main types of tapeworms each of which comes from a different intermediate animal host It has been suggested that they have religious connections the fish tapeworm *Diphyllobothrium latum* being seen more in Jews the pork tapeworm *Taenia solium* in Roman Catholics especially Italians and the beef tapeworm *Taenia saginata* in Protestants Fish tapeworms are from 25 to 30 feet long pork 6 to 12 feet beef 15 to 20 feet The dwarf tapeworm *Hymenolepis nana*  $\frac{1}{8}$  to  $\frac{1}{4}$  inch in length found more frequently in warm climates as in our southern states requires no intermediate host although it is also found in rats and mice

### Symptoms

There are usually no gastrointestinal symptoms except occasionally in children when abdominal discomfort may occur At times children de

for the colex or head. Pouring out the contents on a black plate and teasing out the worm with a probe help in finding the tiny head. If it is not found it may have been lost, may have been killed but not expelled or may still be present and active in the bowel. If it is still active proglottids will begin to be expelled in two or three months when the treatment will have to be repeated.

### Prognosis

In intestinal infestation the results are usually excellent although in 10 or 15 per cent of cases the treatment will need to be repeated. In somatic teniasis the cysticerci may have to be removed surgically if accessible. If they are in vital organs the situation is serious.

### TAENIA ECHINOCOCCUS

*Taenia echinococcus* is a minute tapeworm which lives in the small intestine of dogs, wolves and rarely cats. Man and even horses, sheep and even dogs become intermediate hosts on swallowing the ova which are expelled with the dog's feces. Children are most frequently infested by direct contact with infested pet dogs, but ingestion of food or water fouled by dog's feces can also be a source. Echinococcus disease therefore occurs mostly in countries where dogs are helpers in sheep raising. In this country we see it mostly in Italians.

The shell of the ingested ova is digested by the digestive juices freeing the embryos which enter the portal or systemic circulation. They are carried to various organs and tissues where they become encysted and are known as *hydatid cysts*. These cysts develop a tough wall and have an inner germinal layer producing successive generations of cysts. They may live in the tissues for many years. It is felt that hydatid cysts discovered in the adult probably originated in childhood. They occur principally in females. The organs most frequently infested are the liver (in fully three quarters of cases), other abdominal organs, lungs, nervous system and circulatory system. Hydatid cysts of the liver are discussed in the chapter on the Liver (p. 518). Cysts in the lungs may produce inflammation, gangrene, hemorrhage, perforation and cavity formation resembling tuberculosis. The kidneys become greatly enlarged, the brain may show signs of tumor. Secondary infection may occur. Rupture of the cysts will produce widespread colonies as well as leakage from aspiration or operation.

### Diagnosis

In patients with large tumor masses and an eosinophilia hydatids should be suspected. Symptoms, laboratory findings and x-ray findings will indicate tumors of and damage to specific organs. The development

by an expert. In the stools the ova can be recognized by special techniques. In suspected cases stools after catharsis should be sent to a reliable parasitologist for diagnosis.

### Treatment

*Prophylaxis* is important. All tapeworm carriers should be treated until the worm and its scolex or head have been expelled and preferably burned to avoid spreading of the ova. Care in handling of the segments is important. Meat inspection must be thorough. The larvae known as meaisles are usually found in the jaw muscles of the animal. Thorough cooking of all meat will destroy all larvae.

*Specific treatment* should include the pretreatment care prescribed at the beginning of this chapter. Saline purges each morning and a non-residue low fat but nutritious diet for two or three days before the treatment help to assure success. If the patient is not in good condition he should be given an adequate balanced diet with vitamins and minerals especially iron for a few weeks before he is given the poison to kill the worm.

For many decades *oleoresin aspidium* (male fern) has been used as a vermicide. Many other drugs have been tried none being as good as the male fern although Atabrine has been hailed by some as better. Aspidium is an irritant to the mucosa and when absorbed as when given with oils or alcohol is a poison. Atabrine (quinacrine) also causes severe symptoms with persistent vomiting. Opinion is still divided as to which is more effective. After proper preparation either drug is given in the morning after an all night fast. The oleoresin of aspidium is given in a dose of 4 to 8 gm (1 or 2 drams) usually in four or six capsules one every fifteen minutes. The Atabrine is administered in a dose of 1 gm in ten 0.1 gm tablets two tablets with one half teaspoonful of sodium bicarbonate every ten minutes. One or two hours after the last dose of either drug the worm is washed out by giving 2 ounces of sodium sulfate.

In many cases when a duodenal tube can be successfully passed and identified by fluoroscopy as being in the duodenum either drug can be given through the tube. An emulsion of 8 to 10 gm of aspidium with mucilage of acacia and water 60 cc of each or a solution of 1 gm of Atabrine in 100 cc of water is injected slowly at body temperature. In twenty minutes the bowel is emptied of poison and worms by means of a slow warm transduodenal lavage of 500 cc of a solution of sodium phosphate and sulfate 5 per cent of each (p. 49). The duodenal method obviates some of the bad effects and definitely controls the expulsion of the worm and poison.

The entire contents expelled by either method must be passed into a vessel which is taken immediately to a laboratory for thorough search.

### Symptoms

The symptoms vary with the degree of infestation with the stage of the disease and with the patient's reaction. In the early stages with only moderate numbers of parasites acquired the symptoms may be so mild as to be overlooked. Later severe symptoms develop. In the *stage of penetration* of the cercariae there may be more or less itching called "swimmers itch." There may also be allergic manifestations including urticaria, angioneurotic edema, enlarged lymph glands, mental symptoms and diarrhea. In the pulmonary passage asthma, bronchitis or bronchopneumonia may occur. Eosinophilia and leukocytosis are usually present. A *latent stage* follows while impregnation and ovulation take place. During this time there are usually no symptoms.

During the stage of deposition and extrusion of eggs into the intestinal

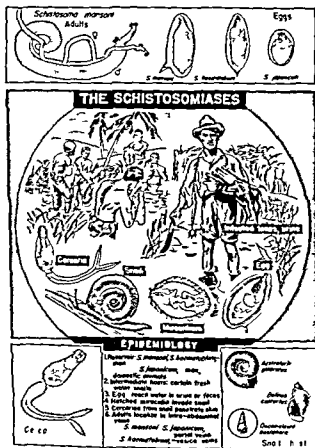


Fig 617 Ep dem ology of the schistosomases (Mackie Hunter and Worth Manual of Tropical Medicine 2nd ed )



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of complications such as hemorrhage infection or rupture may indicate the need for study. A thrill on percussion over a cyst is suggestive.

*Specific tests* are lacking. Precipitin and complement fixation tests have been described but even in most skilled hands are only about 50 per cent accurate. A reliable skin test is now available.

### Treatment

*Prophylaxis* as with all parasites is most important.

Sanitation and avoidance of contact with dogs in endemic areas are necessary. No specific therapy is available. Surgery may be required.

### TREMATODES

Trematodes also known as flukes or flatworms, are seen in this country only in persons who have lived in the tropics where these worms abound such as our service men or travelers who have migrated from such areas for example the Puerto Ricans, Egyptians and Filipinos. The flukes attacking man are of four varieties: blood, liver, intestinal and lung flukes. The most important are the blood flukes or scistosomes.

### SCHISTOSOMIASIS

Schistosomiasis is produced by three kinds of flukes: *Schistosoma mansoni* found in Puerto Rico, South America and Africa and *S. japonicum* found in Japan, the Philippines and nearby countries, both of which cause lesions in the gastrointestinal tract and other viscera and *S. haematobium* which enter the urinary tract and cause hematuria. Man is the definitive host; certain species of snails or mollusks acting as intermediate hosts. As no mollusks in this country are as yet known to be invaded by the flukes, the infestation does not spread here. Recently a Mexican snail has been suspected.

The life history of the parasites is interesting. Man becomes infested when the fork-tailed motile larvae called cercariae penetrate the unbroken skin of a man swimming or working in infested waters. Entering the venules they are carried to the right side of the heart and thence to the lungs. In the lungs they penetrate into venules and are carried to the left side of the heart and thence into the general circulation. Only those reaching the portal vein can survive and become adults. The females there become fertilized, travel against the portal current and deposit their eggs in the mucosa of the intestine or bladder according to the species. The eggs are extruded through the mucosa into the lumen of the intestine or bladder and are excreted. If in or near water they soon hatch into ciliated larvae miracidia which enter suitable snails and are changed into the cercariae. The whole cycle requires at least six to eight weeks.

*Skin and complement fixation tests* are reliable in expert hands and can be used to some extent for screening purposes but biopsy should not be overlooked unless the patient has definite liver damage and impaired blood coagulation which would be a contraindication

### Treatment

*Prophylaxis* is most important in endemic areas but in this country with no intermediate host it is not so important. Patients should be rid of their parasites excreta should be carefully destroyed and any contact with infested waters should be avoided

*Drug Treatment* Antimony has always been recognized as the most effective drug. Tartar emetic given intravenously was used for years but has been superseded by *Fuadin* or *Stibophen* a trivalent antimony compound which is given intramuscularly. The total dosage recommended has varied from 40 to 150 cc of 6.3 per cent solution given in divided doses preferably over not too short a period. When it is given over a five-day period serious toxic reactions may occur including fever severe gastrointestinal and allergic symptoms polyarthritides lung and liver damage and convulsions. The safest total dose is probably 100 cc given in daily doses of 5 cc intravenously for twenty days. It is considered that if one half of cases are cured the treatment has been successful. The treatments must be repeated once or twice if evidence of persistent infestation is present.

*Untoward reactions* are best treated by giving BAI (British antilewisite) 15 cc of 10 per cent solution intramuscularly every four hours for twelve to twenty four hours. This will usually give prompt relief and does not impair the effect of the *Fuadin*.

The chronic abscesses which are complications or sequelae should be treated in the usual way. Central nervous system involvement may require tartar emetic intravenously.

### Prognosis

The chronic fibrotic changes are usually irreversible. The earlier the case is treated the better the result.

### LIVER FLUKES

Liver flukes include several hermaphroditic trematodes that live in the bile ducts of the host and cause liver damage. Fortunately they are rarely seen in this country. They also require passage of their larvae through snails before attacking the human being. The two best known are the *Clonorchis sinensis* endemic in the Far East and *Fasciola hepatica* found in cattle raising countries. The *Clonorchis* requires a secondary host the raw flesh of which when eaten by man produces the infestation. The *Fasciola cercariae* encyst and either float free or are

mucosa diarrhea often alternating with constipation occurs blood mucus and parasite eggs appearing in the stools In *S. haematobium* infestation similar findings take place in the urinary tract There may be fever an increase in leukocytosis and eosinophilia and tenderness in the epigastrium and over the enlarged liver and spleen may be marked As the eggs become lodged in the intestinal wall liver and spleen they may show granulomatous changes about them or may form minute abscesses Gradually with the general weakening of the patient with anorexia epigastric fullness and distress and pain the blood picture changes Leukopenia thrombocytopenia and secondary anemia supervene

The third chronic stage gradually follows The liver becomes fibrotic and symptoms of advanced hepatic cirrhosis may dominate the picture The spleen may be primarily or secondarily enlarged The intestinal wall or with *haematobium* infestation the bladder wall may become fibrotic and papillomas may develop and become malignant Similar changes may also occur in the heart brain and lungs

### Examination

The advanced case shows pallor and evidences of malnutrition The liver and spleen are usually enlarged In the young there is mental retardation The blood findings have been mentioned With hepatic cirrhosis all the findings described in the chapter on this subject (p 506) may be found to a greater or lesser degree Heart lung and urinary changes are to be looked for

### Diagnosis

Awareness is most important If a person has been in endemic area especially bathing or going barefoot parasitic infestation is to be suspected An eosinophilia increases the suspicion In such patients results of x ray and various function studies indicating any of the complications referred to must not be accepted as proof of a primary disease Schistosomiasis must be ruled out

*Stools and urine should be carefully studied for the presence of ova* but these are often missed The finding of concomitant parasitic infestation such as protozoa or metazoa must not be allowed to lead one astray *Proctoscopy* will often show an abnormal looking mucosa sometimes with small granulomatous areas and scrapings may reveal the ova of the flukes The best method of making a diagnosis is by *biopsy* the removal of two or more small areas of mucosa from the first rectal valve about 10 cm above the anal orifice These specimens rinsed and placed between two slides and examined microscopically will usually show the characteristic hooked ova Fixed and stained specimens may reveal granulomas with enclosed ova or shells of ova Repeated biopsies are occasionally necessary

the diaphragm and go through the pleura into the lung where the hermaphrodite adult worm is developed. Rarely these organisms will get to other tissues especially muscles and skin. A few cases have been reported in which foreign body granulomas developed in the abdominal wall producing symptoms and signs resembling those of acute appendicitis. Excision of the granulomatous mass usually cures this situation although of course the pulmonary involvement may go on. Not always ova or even the flukes will be found in the sputum or feces or both. This type of infestation should be borne in mind in treating Asiatics and soldiers who have served in Korea and the East.

### Myiasis

Intestinal myiasis the invasion of the intestine by maggots the larvae of flies is unusual. The skin and various orifices of the body are more frequently the site of this infestation. It is caused by the ingestion of food in which the gravid fly has implanted ova. These ova and even the resulting larvae may rarely survive passage through the stomach and remain attached to the mucosa of the bowel as long as they live usually not longer than a few weeks. As no propagation takes place when all are expelled the infestation is ended. Meanwhile temporary gastrointestinal disturbances with diarrhea but rarely with blood may occur. In small children the symptoms may be violent.

*Diagnosis* is made by the finding of maggots crawling out of the anus often setting up a marked pruritus so that pinworms are suspected.

### Treatment

*Prophylaxis* consisting in the sanitary measures recommended for all parasitic disease is most important.

With no additional ingestion of infested foods the condition is self limiting and not serious. A saline purgative or even a course of hexyl resorcinol as for *Enterobius* infestation may be helpful.

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deposited on aquatic vegetation the ingestion of which in the raw state causes the trouble. *Fasciola* produce more severe allergic symptoms at the onset both flukes cause diarrheas and eventually hepatic cirrhosis, with its typical symptoms

*Diagnosis* is made by finding typical ova in duodenal contents or feces

### Treatment

Prophylaxis is most important as described for other parasites. No specific drugs are known but antimony compounds and gentian violet have been used in *Clonorchis* infestation. For *Fasciola* emetine has been used as in amebiasis.

### INTESTINAL FLUKES

Intestinal flukes invade both large and small intestines and are of various kinds. None are of importance in this country except that an occasional infestation may occur in service men or travelers returning from the East and Near East. Examples are the *Fasciolopsis buski*, *Heterophyes* and *Echinostoma*. All require a snail as the intermediate host and enter the human host through the eating of either raw fish infested with larvae or raw plants containing encysted forms.

*Symptoms* depend upon the degree of infestation. They consist of diarrhea with mucus and occasionally blood and the consequent dehydration. In some instances other tissues are involved including heart, brain and spinal cord.

*Diagnosis* is based on a history of exposure and finding of the ova in stools. The ova must be carefully differentiated from those of tape worms and hookworms.

### Treatment

*Prophylaxis* is the same as for other flukes.

Various anthelmintics are used, none more than 50 per cent effective. Hevylresorcinol used as in *Enterobius* infestation is the most effective and the safest.

### LUNG FLUKES

Lung flukes are of similar characteristics but lodge in the lungs and need not be discussed in a book of this sort except that one type *Paragonimus westermani* endemic in Korea and the East will at times involve the abdomen. The eggs of these flukes are usually passed in the sputum or feces and develop in a fresh water mollusk or in fresh water crabs or crayfish hosts. When the infested hosts are eaten the encysted larvae escape when the cyst wall is digested, penetrate the wall of the jejunum and enter the peritoneal cavity. The parasites then penetrate

## SECTION III

# Diseases of Different Parts of the Gastrointestinal Tract



# The Esophagus

## General Discussion

The two most important primary functions of the body are the ingestion of food and the inspiration of air and nature has provided tough disease resistant organs to carry out these functions. The esophagus is subject to daily trauma from the ingestion of foods of varying consistency more or less masticated and at times containing rough or even sharp admixtures such as cartilages bones shells irritating substances such as spices condiments alcohol tobacco juice drugs and chemicals and even foreign bodies such as pins tacks coins silverware toilet articles and false teeth. In spite of all this abuse the esophagus is comparatively rarely subject to disease recovers quickly from injuries and continues to function under most adverse circumstances even when its lumen is partially obstructed. Because of this fact although the symptoms of esophageal disease are quite characteristic the diagnosis is frequently overlooked.

## ANATOMY AND PHYSIOLOGY

The esophagus at rest is a collapsed tube lying in the posterior mediastinum in contact with the trachea bronchi heart and large vessels and the pleura. The distance from the incisor teeth to the cardia is 16 inches (40 cm) varying little with the size of the individual. Its mucosal lining is of the stratified squamous variety its outer coat is connective tissue merging with its surroundings. There is no serous coat and the muscular coat is strong but flexible so that there is nothing to prevent distention from large food bolus. In recent years there has been considerable controversy over various sphincter mechanisms in the lower esophagus. An inferior esophageal sphincter has been at times demonstrated at or a little above the diaphragm. A slight dilatation above this has been called the ampulla. Below it a slight dilatation extending to the constrictor cardiae is called the vestibule. None of these are invariably found and therefore the practical clinical observation of esophageal physiology will be described. When a bolus is swallowed it passes through the pharynx over the epiglottis produces relaxation of the sphincter like mechanism at the junction of the pharynx and the esophagus and enters the esophagus distending it just enough to permit passage of the bolus and at the same time allow peristalsis to carry it down to the cardia which takes five or six seconds. The cardia normally closed by a sphincter like action of its musculature the constrictor cardiae aided by the





*Cough* usually due to regurgitated contents inhaled into the trachea and often associated with pulmonary complications may frequently be treated for some time before its cause is suspected. The brassy cough resulting from recurrent laryngeal nerve involvement in upper esophageal neoplasm is associated with the characteristic hoarseness due to vocal cord paralysis. Cough is also associated with the serious complication of fistulation into the trachea or the bronchi the sputum often containing food particles in such cases.

*Loss of weight* with later marked emaciation is to be expected from the patient's inability to ingest sufficient food and the resultant symptoms of various food deficiencies may frequently mask the real primary cause.

Other less frequent symptoms of esophageal disease include belching due to swallowing of air in an effort to push down the food rarely to fermentation in long retained esophageal contents various psychiatric manifestations due to frustration in swallowing and a variety of reflex symptoms. Hemorrhage and perforation are complications much to be feared.

#### DIAGNOSIS

It is a fact that early diagnosis of esophageal disease even though the symptoms are so characteristic is rarely made. The dysphagia is mistaken for gastric distress the regurgitation for vomiting the pain for a symptom of other gastrointestinal or of cardiac disease the cough for respiratory disease the emaciation for cancer tuberculosis or neurosis. Careful questioning, as to the relation of symptoms to swallowing should obviate such errors. Esophageal disease should be suspected when any one of the five symptoms mentioned is present and should be considered certain if the first three occur at the same time.

#### Swallowing Tests

A simple swallowing test is of general help. Place a stethoscope high in the epigastrium and have the patient take a swallow of water. The gurgling sound of the water entering the stomach should be heard within ten seconds. If delayed it is suggestive of esophageal narrowing. Determination of the exact nature of the lesion is made by the use of three methods which supplement each other.

*Tubes* The passage of a soft flexible stomach tube is preferable to the use of a bougie which formerly was much used. A regular Ewald tube passed gently into the esophagus may encounter an obstruction at any point. Only gentle pressure should be used in attempting to pass such a point of narrowing. In cardiospasm the tube may at times suddenly pass onward and enter the stomach. If the obstruction is permanent its distance from the teeth may be accurately gauged by compressing the tube firmly by the fingers at the dental line removing the tube and

pinchcock action of the diaphragm through which the esophagus passes or by the inferior esophageal sphincter causes a momentary delay in the passage of the bolus. It opens promptly however so that clinically the entrance of food into the stomach can be heard through a stethoscope over the stomach area within ten seconds of the swallowing act and this can be used to determine whether there is undue delay which would indicate disease. As soon as food enters the stomach the cardia is closed again. It opens only upon the arrival of more ingested material except in cases of regurgitation from the stomach of food gas or swallowed air when the strength of a reverse peristalsis in the stomach overcomes the sphincter action. Such *regurgitation* causes distention of the lower esophagus which in turn causes pain or *heartburn* independent of the content and contrary to popular opinion not due to hyperacidity. Chester Jones produced heartburn by inflating with air a small rubber tube in the lower esophagus the heartburn disappeared upon deflation. If reverse peristalsis in the esophagus occurs its contents regurgitate into the pharynx and mouth. The principal difference between regurgitation and vomiting lies in the fact that regurgitation is mainly accomplished by reverse peristalsis in the esophagus whereas in vomiting the gastric contents are carried by forceful reverse gastric peristalsis all the way to the mouth with little or no help from reverse esophageal peristalsis.

#### SYMPTOMATOLOGY

Because of its simple form and function all diseases of the esophagus result in a group of symptoms which vary in degree and in timing but are present in all. The three cardinal symptoms are dysphagia regurgitation and pain the two most common secondary symptoms are cough and weight loss.

*Dysphagia* may be intermittent due to spasms and temporary or in complete obstruction or constant due to complete obstruction as in the case of congenital anomalies. This difficulty in swallowing is often mistaken for "gas". Occasionally it is encountered in coronary artery disease as a substitution phenomenon for the anginal pain caused by myocardial anoxia.

*Regurgitation* due to partial or complete obstruction characteristically consists in bringing up food which not having reached the stomach does not show evidence of gastric digestion or acid taste milk showing no curdling except in cases of incomplete obstruction associated with regurgitation from the stomach.

*Pain* is a frequent symptom during the swallowing act it is less frequent when the esophagus is at rest. The location of the pain is in the sternal or high epigastric region usually almost exactly over the site of an obstruction or spasm.

*Cough*, usually due to regurgitated contents inhaled into the trachea and often associated with pulmonary complications may frequently be treated for some time before its cause is suspected. The brassy cough resulting from recurrent laryngeal nerve involvement in upper esophageal neoplasm is associated with the characteristic hoarseness due to vocal cord paralysis. Cough is also associated with the serious complication of fistulation into the trachea or the bronchi, the sputum often containing food particles in such cases.

*Loss of weight* with later marked emaciation is to be expected from the patient's inability to ingest sufficient food and the resultant symptoms of various food deficiencies may frequently mask the real primary cause.

Other less frequent symptoms of esophageal disease include belching due to swallowing of air in an effort to push down the food, rarely to fermentation in long retained esophageal contents, various psychiatric manifestations due to frustration in swallowing and a variety of reflex symptoms. Hemorrhage and perforation are complications much to be feared.

#### DIAGNOSIS

It is a fact that early diagnosis of esophageal disease even though the symptoms are so characteristic is rarely made. The dysphagia is mistaken for gastric distress, the regurgitation for vomiting, the pain for a symptom of other gastrointestinal or of cardiac disease, the cough for respiratory disease, the emaciation for cancer, tuberculosis or neurosis. Careful questioning as to the relationship of symptoms to swallowing should obviate such errors. Esophageal disease should be suspected when any one of the five symptoms mentioned is present and should be considered certain if the first three occur at the same time.

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measuring the distance from the tip to the fingers. It is important before doing the measurement to empty into a white dish the contents at the tip of the tube held there because of the finger compression. If the material is bloody, cancer is usually the cause although of course a peptic ulcer might show the same finding. A paraffin block of this bloody material will often disclose particles of cancer tissue, thus confirming the diagnosis. A smear for cytologic study can also be taken.

A small tube preferably the metal tipped flexible *Rehfuss tube* passed for the purpose of doing a gastric analysis may at times show an unusual delay in passing some particular point or may fail to pass it. In cancer suction will produce bloody fluid, which should be examined for cancer tissue or cells. I have found this at times unexpectedly in patients who had as yet no symptoms of esophageal disease. This tube if stopped at the cardia should be quietly left in for twenty minutes or more since if the stoppage is due to spasm the tube may suddenly drop into the stomach.

**X-ray Examination** X-ray study of the esophagus should not only be carefully carried out if symptoms suggesting esophageal disease are present but also should be part of all gastrointestinal, chest and cardiac examinations. As I have pointed out the symptoms of esophageal disease are often misinterpreted as those of cardiac, respiratory or lower gastrointestinal tract disease. Even in the presence of esophageal disease the routine barium mixture given with the patient standing in front of the fluoroscope may not disclose any abnormality. Plain barium mixed with water may show small defects or protrusions or mucosal irregularities. The demonstration of hiatus hernia usually requires a recumbent position with the patient moved around, hips elevated and occasionally with upward pressure in the epigastrium. Diverticula often may not be recognized unless careful search for a residual localized barium accumulation is observed after the meal has passed beyond that point. A thick mixture of barium with a gum or cereal may disclose partial obstruction not seen with the liquid meal and capsules filled with bismuth subcarbonate may be seen to stop at such a point. All these media should be used.

To get good diagnostic films of the esophagus it is usually necessary to take them during the swallowing act in order to bring out the lesion although in cases of complete obstruction the whole esophagus above it can be seen filled with the opaque medium. The site of the obstruction or delay and the appearance of the esophagus above this point can be carefully studied in the films. The irregular defect with moderate dilatation above caused by cancer, the smooth tentlike obstruction with the marked dilatation above caused by cardiospasm, the location, size and mucosal outline of an ulcer or a diverticulum, the thickness and stiffness of the esophageal wall in esophagitis and scleroderma, communication

with the bronchial tree in cases of fistulas or the nature and position of a foreign body—all are characteristic of the particular condition. However the actual nature of a lesion can best be recognized by biopsy. Therefore the third method of examination is always desirable as a supplement to the first two methods.

*Esophagoscopy* Esophagoscopy should never be performed until after x ray study has ruled out the presence of an aneurysm. The esophagoscope should always be passed with the greatest care to avoid not only injury to varicosities but also perforation especially at the site of an ulcer, a diverticulum or an injury. In general the passage of the rigid instrument should also be avoided in the presence of marked dyspnea from any cause and after recent hemorrhage (except when varices are suspected as the cause and the diagnosis must be established).

The passage of the esophagoscope has been done in different positions by different operators. supine, lateral and sitting positions have been recommended. The esophagoscope is a rigid tube and the line of its passage must be a straight line with the patient's head bent backward as far as possible. The length of the upper front teeth determines the degree to which the head must be bent backward, a patient without these teeth being the easiest to examine. Usually a sedative is given before the examination and the pharynx is anesthetized with topical applications to overcome the vomiting and coughing reflexes. In the hands of an expert and patient endoscopist one who is willing to abandon the examination and try again another day if there are circumstances which impede the passage of the instrument, esophagoscopy is a fairly safe procedure although at times even the most skilled examiner will perforate the esophagus. Formerly perforation with resulting mediastinitis accompanied by skin crepitation due to subcutaneous emphysema was an almost invariably fatal complication but today with antibiotics available early operation may be successful in closing the perforation. The advantage of esophagoscopy is that a lesion can be seen and the diagnosis can be made by direct visualization and by biopsy. In cardiospasm the characteristic sphincter like contraction with momentary periods of relaxation can be clearly observed. Another advantage of esophagoscopy is that in some cases treatments can be given through the instrument at the time of the examination.

### Summary

It is of the utmost importance to realize that in any patient with symptoms of esophageal disease the symptoms may be caused or simulated by disease elsewhere and even in the case of any real esophageal lesion there may be other contributing or complicating conditions in the gastrointestinal tract, chest or heart. It is therefore wise never to confine the

study to the esophagus alone, but to have a complete gastrointestinal study in all cases and usually also cardiac and chest surveys

Although with these three methods of diagnosis the nature of the esophageal disease can usually be established additional studies must include examinations to determine the degree of anemia dehydration electrolyte imbalance and food and vitamin deficiencies caused by failure of ingestion of sufficient food and liquids Serological or immunological tests are frequently indicated

### TREATMENT

Although certain procedures are specific for the treatment of esophageal disease it is usually necessary often even before making efforts to establish the diagnosis to institute measures to overcome or control the serious secondary effects of esophageal obstruction

#### Preliminary Treatment

1 Passage of a soft rubber tube to determine the location of the obstruction can be combined with lavage through the tube removing as much of the content of the esophagus as possible and then instilling or having the patient swallow a few ounces of liquid and observing whether it will go through in a few minutes The rate at which it goes through is a guide to the amount of fluid the patient can be fed by mouth Feedings will have to be liquid and can consist of mixtures of milk cream glucose and gelatin with added vitamins mixtures of commercial preparations containing a balanced formula or various combinations of milk eggs meat fruits and vegetables ground up in a blender with added glucose vitamins and minerals (see Liquid Diet p 271)

2 Dehydration and starvation also call for parenteral feedings marked anemia calls for transfusions The feedings given intravenously should contain sufficient protein (amino acids) dextrose and even fat and adequate vitamins and minerals and should not be given to excess being guided by careful observation of the electrolyte balance From 2000 to 3000 calories a day can thus be given It is not necessary today to write out in detail exact formulas for such intravenous feedings since a number of drug houses are supplying bottles containing all necessary ingredients packaged complete with sterile tubing and needles for such injections

3 To prevent aspiration of infective material badly infected teeth especially if pyorrhea is present should be extracted If the teeth happen to be upper front teeth their removal will facilitate esophagoscopy Nose and throat infections should be cleaned up as much as possible

4 Other coexisting conditions should be attended to at once A cardiac patient may require digitalization a diabetic may need careful regulation of insulin dosage a syphilitic may need antiluetic treatment

### Specific Treatment

Specific treatment of the esophagus includes such procedures as dilatation by bougie by a weighted or an inflatable bag or by a branched dilator insertion of a tube through the narrow area and operative procedures. The indications for each form of treatment will be discussed under each disease.

*Surgical treatments* consist essentially of incisions, excisions, repairs and plastic operations. Most of them take considerable time and require a surgeon especially trained in thoracic surgery and an anesthesiologist with experience in this type of work. In other words, teamwork is essential to success. Thorough preparation for operation is of the utmost importance. A complete survey of the patient's condition has already been mentioned as essential in making a complete diagnosis and is even more important when surgery is contemplated. Thoracic cardiovascular, renal, gastrointestinal and other pathological conditions require careful evaluation as to indications for or contraindications to operation. Adequate nutrition should be attained by means of oral and, if necessary, parenteral feedings, as outlined above.

*Postoperative treatment* must be carried out in great detail. General supportive measures started before and during the operation must be continued. The surgeon must supervise the necessary measures to attain and maintain adequate wound drainage and aeration of the lungs. Parenteral feedings must be started at once and as early as possible, supplemented by oral feedings at first of liquids as before operation and later with gradual additions as indicated. X-ray studies, not only of the esophageal function but of the pulmonary and cardiac status, also should be done before the patient is discharged. Follow up at frequent intervals is an important element of successful surgery.

### PROGNOSIS

Operations on the esophagus do not always result in good function. Patients may complain of pain, dysphagia and regurgitation as bad as or worse than before operation. Careful study may disclose cicatricial contraction of the scar producing distortion or stenosis which may require repeated dilatations and perhaps reoperation. In other instances more or less relaxation may occur which interferes with peristalsis and may be difficult to treat even by operation. When attempts have been made to fasten a short esophagus below the diaphragmatic hiatus, it will usually tear loose, producing increased herniation.

### Anomalies of the Esophagus

#### CONGENITAL ANOMALIES

*Absence* of the esophagus or complete *atresia* or an esophagus represented in whole or in part by a fibrous cord produces immediate



regurgitation of feedings and must be recognized at once. Passage of a small rubber catheter will locate the point of obstruction or a swallow of barium will demonstrate it by x ray. Even a plain scout film may show air shadows demonstrating the condition.

*Fistulas* between the esophagus and the trachea or bronchus are usually associated with atresia and will cause not only symptoms of obstruction but also choking, coughing, cyanosis and congestion followed by bronchopneumonia. A scout film may show excessive amounts of air in the stomach and intestines, giving evidence of another fistula between the trachea and esophagus lower down. Barium may be seen in the respiratory tract first and then entering the lower esophagus through the second fistula. Even in atresia without fistulas, however, regurgitated barium (and food) will enter the larynx. The only treatment is prompt gastrotomy with closure of the cardia to prevent such regurgitation. Fortunately for the infant, these lesions are frequently associated with other congenital malformations, such as imperforate anus, cardiac anomalies and others, so that there is practically no hope for survival.

*Congenital fibrous narrowing* of the esophagus, if causing complete obstruction, is classed with atresia. Moderate degrees of narrowing may cause only occasional regurgitation at first, becoming more frequent as solid foods are added to the diet of the child and requiring dilatation in the same manner as stenosis in the adult. Occasionally a congenital narrowing is encountered in the adult who has accustomed himself since childhood not to attempt to swallow too large bolus of food because of the knowledge that these will stick at the point of narrowing and cause much distress in efforts to get them up, or he will force them through with repeated swallowing of water, bending of the body, coughing or even assistance with the finger in the throat. At times removal of a mass of food may have to be done by a physician, preferably one skilled in endoscopy. Careful x ray study will disclose the narrowing and its nature will be confirmed by esophagoscopy. Dilatation is indicated as in cicatricial stenosis from other causes.

*Congenital webs* or bands usually occur in the upper part of the esophagus and may, when lying flat against the mucosa of the esophagus, cause no symptoms, but when protruding into the lumen may act like the cusps of valves, causing food to accumulate above them, thus causing obstruction. They are usually accompanied by more or less fibrous narrowing. These bouts of temporary obstruction occur intermittently and usually come on suddenly, often in the middle of a meal. The patient will leave the table, go through the same contortions as with the stenosis just described, get out the bolus of food and resume eating without further discomfort. This history of intermittent dysphagia and the loud gurgling sound often noticed when such patients swallow liquids resembles that of a pulsion diverticulum. The finding of a fine membranous



Figure 18 Congenital web of esophagus (patient's age 35) (1) fibrotic narrowing (2) dilatation (3) congenital web

appearing defect with some narrowing on careful and repeated x ray studies (Fig 18) will help in the diagnosis which is confirmed by esophagoscopy. The treatment consists usually in simple cutting or tearing of the web through the esophagoscope and one such treatment usually suffices to eliminate this annoying condition.

### DIVERTICULA

Diverticula are of three types—the so called pulsion, traction and traction pulsion varieties.

#### PULSION DIVERTICULUM

This diverticulum of the upper end of the esophagus really at the junction of the pharynx and esophagus is also known as Zenker's pouch. It is really not a true diverticulum but a protrusion of mucosa and submucosa through the muscular coat usually posteriorly and to the left which is due to congenital weakness at this point and pressure of the food bolus against it. The entrance from the esophagus is small so that only small amounts of liquid food can enter it. Over the years the pouch grows larger and may eventually cause enough pressure upon the side of the esophagus to cause obstruction. When it is small there may be only



Figure 19 a Pulsion diverticulum (1) diverticulum (2) esophagus compressed above by diverticulum b Carcinoma in pulsion diverticulum (1) diverticulum (2) irregular outline of carcinoma

throat irritation later followed by the feeling of a lump and by dysphagia up to the point of the complete obstruction. Usually the patient can empty the pouch by bending far forward and may actually rinse out the pouch by swallowing water and letting it run out repeatedly. The material obtained will of course be the food as swallowed unchanged by digestion. Fermentation produces a very bad odor to the breath. All these symptoms including gurgling on swallowing resemble those described as occurring in cases of congenital cels.

#### DIAGNOSIS

The passage of a *stomach tube* may not be interrupted at all unless the sac has become very large and the lumen distended. Obviously any forceful push on the tube might rupture the diverticulum. Partial withdrawal of the tube and its reintroduction may bypass the lumen and permit the tube to go down to the stomach. *X ray examination* makes the diagnosis. It is preferable to use a thin barium mixture. The filled pouch is clearly seen after the esophagus has emptied itself although at times the pouch can be filled only by having the patient take different positions lying down (Fig. 19 a). By *esophagoscopy* it is usually difficult to disclose the small lumen even when the diverticulum is known to be present and when it is entered there is danger of perforation.

#### TREATMENT

Many patients get along for years by avoiding forceful swallowing of large bolus but particularly of fluids and by simply washing out the sac

with water either regularly after meals or when it becomes uncomfortable. There exists the danger of the chronic irritation causing malignant changes although this rarely occurs. Figure 19 *b* shows a roentgenogram of a patient with adenocarcinoma involving the pouch. At times occasional dilatation through an esophagoscope of a narrowing or spasm below the stomach of the diverticulum by decreasing delay at this point will do much to prevent filling of the pouch. Operative removal of the diverticulum is difficult because it is hard to find it among the complex cervical structures. It can however be easily found if the tip of an esophagoscope is inserted into the sac before the operation and kept lighted to guide the surgeon. Yet even the most careful operation done in two stages may result in development of a fistula through which mucus will drain. This makes the healing process difficult if not impossible. Some surgeons still believe that fixing the diverticulum in an inverted position in the neck so that it will drain into the esophagus instead of receiving its contents is the operation of choice since with no incision into the sac no fistula can develop.

#### TRACTION DIVERTICULUM

Traction diverticula occur at any point below the upper end of the esophagus. They are due to traction on the esophageal wall from inflam-

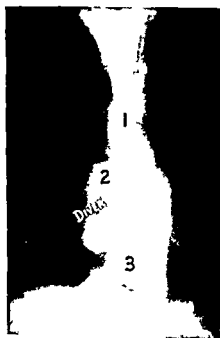


Figure 20 Traction diverticulum (1) esophagus slightly dilated (2) large diverticulum (3) lower esophagus



Figure 19 a Pulsion diverticulum (1) diverticulum (2) esophagus compressed above by diverticulum b Carcinoma in pulsion diverticulum (1) diverticulum (2) irregular outline of carcinoma

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ber of classifications of such hernias exist for all practical purposes there are two types the hiatus hernia which is not uncommon and hernias elsewhere which are much more rare usually traumatic although occasionally congenital

Hernias other than hiatus hernias may be due to *failure of development* of the diaphragm producing either a large congenital opening which will cause death early or a smaller opening or weak area which is converted into a hernia by trauma or other causes producing sudden excessive intra abdominal pressure. These hernias are usually on the left side the liver largely protecting the right side from herniation. Herniation may be to the right however through the foramen of Morgagni. The purely *traumatic hernias* result from penetration of the diaphragm by bullet stab or other wounds which may produce herniation immediately or considerably later when another trauma will act the same way as in the congenital type. Most commonly the stomach and transverse colon will pass upward into the chest but other organs have been observed up to the point of almost complete evisceration into the chest. Such multiple protrusions have been given the name of "compound hernias." Strangulation may occur in the compressed intestines and perforation may produce pleuritis or mediastinitis.

#### SYMPTOMS

The symptoms vary with the location and extent of the herniation. A small hernia with only occasional protrusion upward of part of the stomach or transverse colon may cause no symptoms. When esophagitis and gastritis complicate the situation there may be only gastric irritation, aggravated by eating or symptoms of intermittent partial esophageal obstruction with perhaps some oppression in the anterior chest. Not only will large protrusion cause an aggravation of the gastrointestinal symptoms but also the pressure of the abdominal organs against the heart and lungs may produce embarrassment of their functions resulting in symptoms suggestive of cardiovascular disease of pleurisy with severe chest pains cough and cyanosis which in turn may result in pulmonary congestion and pneumonia. Perforation due to gangrene from strangulation of the bowel will cause sudden severe chest pains suggesting coronary occlusion or pulmonary embolism.

#### PHYSICAL EXAMINATION

In mild cases no abnormal signs may be found in more severe cases there will be signs of left sided diaphragmatic fixation abnormal tympany or dullness in the chest (particularly the left) heart displacement and occasionally an appearance of retraction of the left upper side of the abdomen. Auscultation will show absence or displacement of breath sounds the cardiac displacement and often peristaltic sounds and gurgling.

matory disease contiguous to it usually tuberculosis or mycosis of the lungs mediastinum or spine They may be small or large and may have a lumen that is irregular as shown in Figure 20 They usually cause no specific symptoms distinguishable from the disease causing them although with healing of the original lesion there may be a feeling of fullness in the esophagus Rarely they may cause a mediastinal abscess or a fistula into a bronchus Usually they are found on a routine gastrointestinal roentgen study and no treatment is necessary unless the rare complications occur

#### TRACTION PULSION DIVERTICULUM

*Traction pulsion diverticula occur at the lower end of the esophagus just above the hiatus they are supposed to be due to a combination of*



*Figure 21 Esophagospasm simulating diverticula a Protrusion at side (1) resembling pulsion diverticulum b Two protrusions (1 and 2) c Slight dilatations (1 and 2) with spasm between*

pulsion and traction They may be large or small usually with a wide opening and cause no symptoms They must be differentiated from hiatal hernia on x-ray examination In the extremely rare cases in which obstruction spasm or malignancy complicates this condition surgery may be necessary In spasm dilatation may be satisfactory At times temporary spasm may produce an appearance of diverticulation (Fig 21)

#### DIAPHRAGMATIC HERNIA

Though not strictly an esophageal condition diaphragmatic hernia is usually included in discussions of esophageal diseases Although a num

the hiatus hernia may be the result of congenital weakness or enlargement of the hiatus or it may result from senile changes such as loss of fat decreased elasticity of connective tissue and muscular displacements. Some writers have included as a cause a congenitally short esophagus which pulls the cardiac end of the stomach or even the entire stomach above the hiatus. Others point out that this is not a true herniation. In the true hernia there may be upward protrusion of a small or a large part of the cardiac end of the stomach or in rare cases of nearly the entire stomach (the "upside down stomach"). Protrusion of intestine through the hernia rarely occurs. The protrusion is usually behind and to the left of the hiatus. The stomach may rarely be held in this position but usually, it tends to slide up when the patient is lying down in a certain position and is pulled down when the patient stands up especially if the stomach is weighted down with some content.

#### SYMPTOMS

The hiatus hernia may be present for years never showing any symptoms and only found on routine gastrointestinal x-ray study or when an esophagogram is done during a cardiac study. Mild symptoms may consist in a fullness or pulling sensation in the lower sternal region described as "gas." Rarely a mild intermittent dysphagia may be described. The most characteristic syndrome consists in a substernal precordial distress or pain waking the patient up at night in a state of great apprehension and often accompanied by dyspnea so that a "heart attack" is suspected. However on turning in bed or more frequently when the patient gets up and walks around after taking a glass of water plain or containing some medication the stomach suddenly slips out of the hiatus and symptoms cease abruptly. The patient usually learns to get up and take his medication when the attack occurs giving credit to the medication rather than to the water with which it is swallowed. The water of course acts to weight down the stomach pulling it downward and out of the hernia.

Occasionally an ulcer may complicate the herniation. An ulcer of the esophagus or esophagitis with marked congestion may occur. It may be the cause of much more severe pain than is seen in uncomplicated hiatus hernia and may result in hemorrhage or stricture formation producing dysphagia. I have also seen a few cases in which a gastric ulcer could be seen in the part of the stomach in the hernia the ulcer with the induration preventing the passage through the hiatus so that the stomach remained fixed above. The ulcer in one patient healing in a normal fashion resulted in scarring and probably adhesions because one third of the stomach remained fixed above the diaphragm. The possibility of cancer occurring in the irritated area must be considered. It would cause continuous symptoms and gradually develop into an obstruction.



## X RAY EXAMINATION

X ray examination will show the displacement of heart and lungs and the shadows of the abdominal organs identifiable by the presence of gas in the intestines confirmed by giving a barium meal or barium enemas (Fig 22) If on standing the hernia has reduced itself however nothing may be found to account for the history and symptoms It is important to take the x ray films with the patient lying down in various positions including the Trendelenburg position



Figure 22 *a* Diaphragmatic hernia (1) dilated esophagus (2) most of stomach in chest (3) hernial ring (4) remainder of stomach below ring *b* Diaphragmatic hernia (traumatic) (1) large hernial ring (2) colon in sac

## TREATMENT

The treatment especially when the herniation is extensive is surgical Various procedures have been recommended most of which are satisfactory although scarring may provide a weak area subject to further herniation

## HIATUS HERNIA

Hiatus hernia a common condition found frequently in persons at middle age and older is the name given to several different conditions in which the cardiac end of the stomach is able to slip upward through the esophageal hiatus of the diaphragm into the chest Like hernias elsewhere

be seen until x ray films taken in different positions including the Trendelenburg will show it up clearly (Fig. 23) Films should always be taken and other gastrointestinal conditions which might be causing symptoms should be ruled out by a complete gastrointestinal x ray study

#### TREATMENT

Most cases with no symptoms require no treatment When symptoms are present the condition should be carefully described to the patient with the aid of pictures and the method of obtaining relief by standing and drinking one or two glasses of water should be explained Usually patients will accept the slight or moderate discomfort if they understand the cause and realize that it cannot be corrected except by difficult operative procedures In more severe cases with the stomach fixed above the diaphragm plastic operations upon the wide hiatus with fixation of the stomach below it may at times cure the condition but frequently the patient will be worse with stricture as a serious complication Gastroenterostomy and even gastrectomy have been performed In cases of a short esophagus operations are particularly unsuccessful since the esophagus usually reascends pulling the stomach with it

#### EVENTRATION OF THE DIAPHRAGM

Eventration of the diaphragm from which herniation is at times difficult to distinguish is a prominent elevation of the diaphragm usually to the left side It is usually due to congenital weakness or to insufficient development of the whole muscle permitting the abdominal organs to push the diaphragm upward into the left side of the chest Some cases of eventration may possibly be produced as a result of prolonged pressure against the diaphragm by an abnormally distended stomach or colon In some cases phrenic nerve paralysis may be the cause The abnormally high position of the stomach and colon may produce indefinite gastric and colonic symptoms The pressure against the lung may cause embarrassment or even collapse of the lung By careful x ray study in various positions it is usually possible to demonstrate that no part of the stomach or colon extends above the upper border of the diaphragm

There is no specific treatment for this condition but maintenance of normal gastrointestinal function and avoidance of overdistention will usually make the patient more comfortable

#### CARDIOSPASM (ACHALASIA)

Although temporary spasms of the esophagus at other and often varying parts of the esophagus occur rarely (Fig. 21 p. 182) and may be due to local irritation from ulcer esophagitis or injury or reflex from disease elsewhere the most frequent location of so called spasms is at the cardiac or lower end of the esophagus There is still considerable

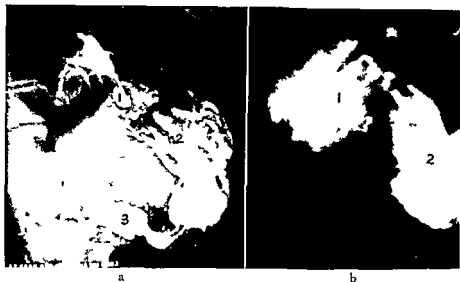


Figure 23 Hiatus hernia *a* Shows (1) ulcer crater in part of stomach above diaphragm (2) remainder of stomach (3) duodenal cap *b* Shows (1) same part of stomach above diaphragm ulcer healed (2) remainder of stomach

### DIAGNOSIS

The typical history of the nocturnal attacks described above should occasion a suspicion that hiatus hernia is the cause. Coronary insufficiency often confused with this condition does not produce the typical cessation of symptoms on drinking and walking in fact the symptoms would be increased by such activity. Both the herniation and coronary sclerosis however may be present in the same patient at the usual age at which these conditions occur. It is therefore important to make a thorough cardiac as well as gastrointestinal study in all such cases.

*Physical examination* does not usually show any characteristic findings in hiatus hernia although it is of course a great help in cardiac cases. As the stomach usually lies posteriorly abnormal tympany would rarely be elicited.

*Laboratory examinations* are of no help although there might be some difficulty in passing a stomach tube when the stomach is above the diaphragm. Usually however as stated before the stomach is in normal position when the patient is up.

Careful *x ray study* is necessary to make the diagnosis. Frequently these hernias are overlooked when the patient is standing for fluoroscopy although a wide cardiac orifice should occasion suspicion. On the other hand a hernia is often mistakenly diagnosed when the lower end of the esophagus is somewhat dilated. Fluoroscopy in various positions standing and lying down with manual pressure on the abdomen at times may be necessary to produce the herniation. In many cases the hernia will not



Figure 24 Cardiospasm (achalasia) showing (1) dilated esophagus (2) smooth tapering obstruction

opportunity to drop into the stomach. Blood in the aspirated material from above or below the spasm suggests the presence of carcinoma.

The x-ray examination shows a more or less dilated esophagus with the lower end tapered almost to a point or occasionally a teatlike tip where it enters the stomach (Fig. 24). There may often be seen a fine line of barium going through the narrowed area at intervals. At times the dilated esophagus may be of enormous size, interfering with breathing and with cardiac function when filled. It is usually necessary to lavage the esophagus before the examination but very gently to avoid perforation.

*Esophagoscopy* also after a lavage except in the early stages discloses the closed lower end which presents a smooth sphincter-like appearance, often with a fine tremor and with occasional momentary opening permitting small amounts of food and even the tip of the esophagoscope to enter the stomach.

#### TREATMENT

Rarely achalasia is a temporary condition and may subside spontaneously, the credit being given to medication or to psychologic or other treatment. Sometimes the mere pushing of a normal diet with food of

difference of opinion as to the exact nature of this condition. Some still consider it a spasm of the sphincter at the cardia; some describe it as due to a ring; some consider that it is part of a general atony and dilatation of the esophagus; and some regard it as a fibrosis of the sphincter muscle. Most authorities today consider that it is not a spasm but a failure of normal relaxation of the sphincter when food should pass into the stomach. At times it has been shown to be due to allergy. Occasionally it follows operations upon the stomach. It is particularly important to realize that cardiospasm may occur as an early sign of cancer high up in the fundus of the stomach long before the cancer actually involves the cardiac orifice and causes real stenosis.

### SYMPTOMS

The symptoms are fairly characteristic and usually occur in middle age or beyond. At first they are intermittent. The patient may suddenly experience a feeling of constriction or fullness in the region of the esophageal process and realize that food, especially liquid, is not passing through this point. With considerable effort, contracting the chest muscles and stooping over, sometimes assisted by swallowing air or a carbonated beverage, there may occur a sudden relaxation and food may be felt going through into the stomach. Attacks of this sort usually occur with increasing frequency and severity; food finally refuses to go through any more and is regurgitated in increasing quantity and at gradually increasing intervals. Eventually there appears to be complete obstruction. The regurgitated food, varying in amount from a few ounces to a quart or more, *looks and tastes unchanged except in occasional instances when a little gastric juice may regurgitate into the lower esophagus.* Large quantities ofropy, tenacious mucus are mixed with the food or may be regurgitated alone. There is usually no evidence of blood unless excessive retching has caused injury to the mucosa or an ulcer is present. The patient becomes emaciated and dehydrated and presents evidences of food deficiencies. The appearance of the patient suggests malignancy.

### DIAGNOSIS

Although the symptoms are characteristic, they somewhat resemble those caused by diverticula, by congenital veils and by benign or malignant stricture. The passage of a large stomach tube, interrupted at the lower end of the esophagus (approximately 16 inches from the teeth), may, after gentle pressure for a few minutes, suddenly drop into the stomach. This is proved by the clear gastric juice obtained on aspiration and is definitely confirmed visually if there is a bile stain to the liquid. A Rehfuß tube likewise will rest above the point of "spasm" and may be allowed to remain there for a half hour or longer in order to give it an

seven healed ulcers in 4000 esophagoscopies an incidence of 2.5 per cent of patients referred to him as problem cases from all over the world. Various causes for the formation of such ulcers have been discussed most of which have been based on the idea that they are caused by digestion of a small area of esophageal mucosa by regurgitated acid gastric juice. This regurgitation may occur as a result of (1) reverse peristalsis from pyloric spasm or obstruction or (2) a wide patulous cardia as in hiatus hernia or short esophagus or (3) after operations on the stomach especially when followed by prolonged intubation. Another cause is supposed to be the breaking down of small islands of aberrant gastric mucosa in the esophagus. Ulcers or erosions may also accompany esophagitis and on the other hand be the cause of surrounding esophagitis. Allergy may also be a cause. The resemblance to gastric and duodenal ulcer has been pointed out their occurrence in patients with such ulcers has been noted and a common etiology has been assumed. They usually occur in the lower third of the esophagus may heal rapidly or may result in such complications as hemorrhage cicatricial contraction with stenosis or perforation either below the diaphragm into the lesser peritoneal cavity or above it into the mediastinum pleura pericardium or lung.

#### SYMPTOMS

Many times scars have been found in patients who had never experienced any symptoms. The characteristic symptom is lower substernal pain occurring during swallowing or immediately afterward at times with solid and not with liquid food. When the ulcer is associated with gastric or duodenal ulcer the symptoms may be indistinguishable. Dysphagia is usually more or less marked being the result of spasm or induration at the ulcer site or healing with cicatricial contraction and more or less stenosis. Regurgitation may occur and if it is a result of esophageal narrowing will be of the usual esophageal type not acid and containing undigested food if due to pylorospasm or stenosis it will be strongly acid with partially digested food content. Hemorrhage is a frequent complication and may cause hematemesis or only *melena* with resulting anemia but rarely death. Perforation will cause the symptoms of either an acute abdominal or of an acute cardiac or pulmonary catastrophe or ruptured aneurysm and as it may occur in patients with no previous symptoms may be impossible to diagnose except at operation or autopsy.

#### DIAGNOSIS

Esophageal ulcer when presenting symptoms may frequently be confused with any other esophageal lesions such as esophagitis cancer or achalasia. When hemorrhage is the first symptom opinions about its origin may vary nasopharyngeal pulmonary gastric or duodenal

firm consistency will relieve the condition. Medication is of no avail although temporary mild sedation may be required until something definitive can be done. The condition not being a real spasm anti-spasmodics and anticholinergics usually have no effect. Treatment must be directed to the restoration of the normal relaxation of the cardia when esophageal contents are pressed against it. At times the mere passage of a stomach tube either the large tube or the Rehfuess tube as mentioned above in describing a method of diagnosis will produce temporary or even permanent relief of the condition. In more severe cases stretching of the cardia is the important thing to be accomplished and should be done by a person who has had experience with this procedure and is cognizant of and equipped to handle the dangerous results of perforation.

Dilatation has been accomplished in many different ways. Some still advocate the passage over a previously anchored silk cord or wire of increasing sizes of olive bougies. Others use soft rubber bougies loaded with mercury or small buckshot which will gradually slide through the cardia. Others pass tubes surrounded or tipped with inflatable bags, some pulling a bag distended with air upward through the cardia, others placing a collapsed bag in the cardia under fluoroscopic control and distending the bag with air or water, the pressure being measured by a manometer. An experienced esophagoscopist may use a branched dilator similar to a Kohlmann urethral dilator. The greater the dilatation the more successful the result but also the more dangerous the procedure. One thorough dilatation may completely cure the condition whereas in some patients repeated dilatations may be required.

Lavage which an intelligent patient can be taught to do himself and the ingestion of emollients such as oils, gelatin or Amphojel may be of value. Surgical procedures include gastrostomy with dilatation of the cardia by the finger or dilators over a previously swallowed string, longitudinal incision with transverse closure similar to the Ramstedt operation for cardiospasm in infants, plastic operations, esophagogastrostomy and simple gastrostomy. The results of operative intervention are not as good as those obtained by skillful dilatation from above. In either case when the condition has existed for a long time and the esophagus has become badly atonic and distended more or less trouble with deglutition can be expected for a long time requiring repeated treatments by lavage and sphincter dilatation. After operation a stricture may develop which may require stretching and in the end a permanent gastrostomy.

## Diseases of the Esophagus

### ULCER OF THE ESOPHAGUS

Ulcer is a rare condition but has been much written about since Chevalier Jackson in 1929 reported finding twenty one active and sixty

an esophagoscope have been made but perforation should be feared from such treatment. The ingestion of bismuth powder after feedings with the idea that it will adhere to the ulcer has the disadvantage of causing constipation. The esophagus may be put to rest by liquid feedings through a small polyethylene tube passed through the nose into the stomach using mixtures of milk cream glucose and gelatin or solutions of one of the commercial nutrient powders (Nutragest). Oil in small amounts should be swallowed to prevent irritation from the tube.

The ulcers usually heal promptly, but persistence of the ulcer may in rare cases require operation. With healing the area should be checked occasionally to determine whether cicatricial stenosis is about to occur, and if so dilatation may be required as in any benign stenosis (p. 198). Hemorrhage calls for the same treatment as hemorrhage from gastroduodenal ulcer (p. 269) but when profuse and persistent prompt x-ray study and esophagoscopy should be done especially to rule out bleeding varices which require immediate treatment. Perforation demands immediate operation.

#### ACUTE ESOPHAGITIS

Esophagitis may be acute or chronic. The acute form is of course present after caustic or irritating substances have been swallowed. It may be due to trauma from swallowed foreign bodies, excessive vomiting, too drastic esophageal instrumentation, prolonged esophageal intubation and to allergy.

The *symptoms* include esophageal burning or pain, regurgitation and occasional bleeding. When inflammation and induration are excessive symptoms of real obstruction may occur. Actual stricture may result from scarring. Exsanguinating hemorrhage has been known to occur.

#### DIAGNOSIS

The stomach tube will pass slowly and with difficulty and extreme caution must be used to avoid perforation. The x-ray film will show spasms and thickening and esophagoscopy will reveal more or less diffuse inflammation, occasionally with erosions or actual ulceration. Biopsy may be done to rule out malignancy.

#### TREATMENT

Treatment should consist in the feeding of emollients such as oils or gelatin solution (one teaspoonful of powdered gelatin in 1 or 2 ounces of warm water) and well balanced liquid foods with added gelatin (see p. 271). The consistency of the feedings can be gradually increased and a bland diet should be continued for a few weeks after the acute episode. In the presence of actual infection antibiotics may be used for a short time. In allergies determination and elimination of the cause are





Figure 25 Ulcer of esophagus showing (1) esophagus slightly dilated (2) ulcer crater lower esophagus (3) fundus of stomach

hemorrhage being suspected as well as esophageal varices or malignancy. Stenosis without previous symptoms will occasion a suspicion of swallowing of caustics, insidious growth of cancer or other causes. Differential diagnosis is often difficult. The stomach tube may simply indicate esophageal bleeding or obstruction.

X-ray examination rarely discloses an ulcer crater (Fig 25) it may show only spasm at the site of an ulcer but will of course demonstrate most of the other diseases mentioned. Esophagoscopy will many times demonstrate the ulcer and a biopsy will usually differentiate malignancy. A scar of an ulcer may be seen at an area of narrowing.

#### TREATMENT

Removal or treatment of the causes mentioned constitutes an important part of the management of these cases. Treatment of the ulcer per se should be that of gastric or duodenal ulcer (see section on ulcer p 266) and a search for allergic factors is important (see allergy p 89). Frequent feedings of bland nutritious foods is indicated preceded by administration of oil or a solution of gelatin one teaspoonful dissolved in 1 or 2 ounces of warm water if narrowing is present. Attempts at treating the ulcer locally by applying 10 per cent silver nitrate through



Figure 26 Cicatricial stricture of esophagus showing, (1) slightly dilated esophagus above stricture (2) stricture due to inflammation (3) fundus of stomach (*magenblase*)

### DIAGNOSIS

Diagnosis must be made by excluding all other types of diseases causing esophageal symptoms. A careful and detailed *history* will frequently elicit the cause of the esophagitis. The *stomach tube* may show the narrowing, if present. The *x ray* films will show a smoothly or irregularly thickened wall with more or less narrowing of the lumen (Fig 26). *Esophagoscopy* with biopsy may establish the diagnosis. The possibility of malignant disease coincident with esophagitis or secondary to it must be borne in mind. The need for a thorough search for the various causes is obvious. blood studies—including serology, agglutination and tuberculin tests—sputum examinations, allergy studies, chest and gastrointestinal x rays and careful search for oral, respiratory and rectal infections should be done.

### TREATMENT

Treatment should consist in removal or suitable treatment of the various causative factors, careful regulation of nutrition, starting with frequent feedings of either liquid or soft diet with added vitamins and iron, and insistence upon proper mastication. If strictures result from

- 194      **Different Parts of the Gastrointestinal Tract**
- necessary    Stricture must be treated as previously described    careful dilatation being started early

### CHRONIC ESOPHAGITIS

The chronic form may be of several different types some originating in the esophagus some secondary to other esophageal conditions or external causes. It may be caused by several factors

1 Habitual ingestion of irritating substances such as excessively hot or cold liquids chemicals irritants such as alcohol tobacco juice from chewing tobacco or cigars spices drugs or insufficiently masticated foods

2 Frequent and prolonged regurgitation or vomiting of hyperacid gastric juice due to gastric disturbances such as gastric or duodenal ulcer associated with pylorospasm and reverse peristalsis

3 A widely patent cardiac orifice permitting regurgitation

4 Peptic ulcer of the esophagus when the esophagitis may be the cause or the effect of the ulcer

5 Nutritional deficiencies due to improper diet and interferences with the blood supply to the esophagus producing chronic stasis and lowering of the resistance of the esophageal wall. This type is seen with esophageal varices

6 Damage which may be more or less permanent may result from acute infectious diseases such as diphtheria scarlet fever typhoid fever and agranulocytosis or from prolonged ingestion of foods to which the patient is allergic (see p 87)

7 Extension from chronic infections in the mouth nose throat trachea and bronchi

8 Specific involvement of the esophagus from tuberculosis of the throat larynx lungs lymph nodes and spine occasionally associated with fistulas

9 Syphilitic involvement in the form of fibrosis ulcers gumma or functional disturbances secondary to cerebrospinal involvement

10 Mycotic infections blastomycosis and actinomycosis rarely primary usually secondary to these infections elsewhere (see p 623)

11 Heterotopic gastric mucosa is rarely the seat of inflammation

### SYMPTOMS

Symptoms may be those of any other esophageal lesion that is sub sternal pain on swallowing some dysphagia and occasional regurgitation which in some cases may be sour. The symptoms of the primary disease can usually be elicited such as the typical peptic ulcer syndrome chronic alcoholism, the history of acute infectious disease or of tuberculosis syphilis or mycotic disease chronic upper respiratory tract infection and symptoms of malnutrition and anemia

## CICATRICIAL STENOSIS

*Intrinsic narrowing* is usually due to cicatricial contraction as a result of more or less extensive necrosis due to the swallowing of caustics. It may also rarely occur as a result of other trauma of peptic ulcer of the esophagus, scleroderma, varices or esophagitis, but in these cases it is rarely as complete as that due to caustics. There is a history of the swallowing of a strong alkali, lye or caustic soda or acid, usually muriatic acid, frequently by accident though occasionally with suicidal intent. It is not seen as frequently today as formerly as a result of an educational campaign calling attention to the danger of leaving alkalis or acids used for washing or cleaning purposes in places accessible to persons, especially children, who are not aware of the danger of drinking anything they see around, even though marked "Poison."

Upon swallowing the caustic the patient has immediate horrible burning of the mouth, pharynx and esophagus with retching so that no great quantity of the stuff is usually swallowed. At times, however, larger quantities may be swallowed as in the case of a hospital orderly who scolded for being drunk and late to work drank a half pint of muriatic acid used for cleaning tile in the hospital. A considerable quantity of the strong acid reached his stomach so that in addition to the usual burns and cicatrices of the esophagus he also suffered a pyloric stenosis.

Suitable neutralization of the acid or alkali in any case followed by the administration of emollients to soothe the irritated mucosa and parenteral feedings to maintain nutrition and electrolyte balance are only the first steps in the treatment of the burns. Within a few days an effort should be made to pass a small tube such as the Levin tube into the stomach, being careful to precede its passage with the swallowing of oil, vegetable or mineral so that the tube will be encouraged merely to float down in the oil. Any pressure on the tube must be avoided because of the danger of perforating the friable esophageal wall. Many times no tube will pass, cicatricial contraction will occur and the patient will be able to swallow only liquids or nothing at all. I have seen patients go for months this way with no attempts at treatment and with the lumen getting progressively smaller even to the point of complete stenosis.

## DIAGNOSIS

The *history* of ingestion of the caustic, the dysphagia and the inability to pass a stomach tube beyond a certain point will make a diagnosis of stenosis which can also be seen by esophagoscopy.

The *x ray* films in complete stenosis will show the total cutting off of the barium column. In incomplete stenosis they will show a smooth narrowing which may extend for several inches or may show more than one such area (Fig. 27). Unless the barium flows all the way into the

prolonged inflammation and ulceration they should be treated as recommended in the section on benign stenosis

### ALLERGY

Sensitivity to foods medications inhalants contacts or bacterial or mycotic infections elsewhere as mentioned in the chapter in Gastrointestinal Allergy (p 87) occasionally will produce manifestations in the esophagus These may take the form of a generalized acute reaction producing edema redness round cell infiltration and possibly superficial erosion or ulcers with bleeding purpuric areas often with bleeding or the picture of an acute esophagitis or esophageal ulcer subsiding rapidly when the cause is removed When the offending causes are constantly present chronic irritation with development of esophagitis or chronic ulcer may occur At times spasms of the esophagus and even achalasia can be shown to be due to allergy For further description of the various conditions mentioned the reader should refer to them under the appropriate headings

The *symptoms* will be those of the condition mentioned Allergy should be suspected when acute esophageal symptoms occur at intervals with no other cause demonstrable and when no definite cause for chronic esophageal lesions can be determined

The *diagnosis* must be made by careful and thorough studies for allergy after organic causes have been ruled out and may be aided by obtaining a history of definite allergic manifestations elsewhere the finding of eosinophilia during acute attacks and by a favorable response to epinephrine ephedrine and antihistaminics At times it may be convincingly demonstrated by x ray study or esophagoscopy when a reaction occurs after the ingestion of a suspected substance or its topical application to the esophageal mucosa Skin tests are of no value

### TREATMENT

Essentially removal of the offending allergens is the only really successful treatment emphasizing that the search for these factors is most important Medication directed to amelioration of symptoms by the use of epinephrine ephedrine antihistaminics or steroids is of no permanent value Desensitization of value in the case of pollens or other inhalants is of no value in allergy to food or medication Eradication of focal infections may eliminate important sources of allergenic products Adequate care of any concurrent disease and proper attention to the patient's nutrition and hygiene are important adjuncts to treatment

### STRICTURES OF THE ESOPHAGUS (STENOSIS)

Strictures of the esophagus are usually classified as benign and malignant each classification being further subdivided into those caused by intrinsic and by extrinsic factors

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sive with difficulty first in swallowing solids then mushy foods and finally liquids Rarely when superficial sloughing occurs there may be a brief respite but even this is unusual Food in varying amounts is regurgitated unchanged and contains much mucus frequently blood tinged At times only a little bloody mucus may be regurgitated during sleep More or less pain may occur in the chest or epigastrium either constantly or only with swallowing The breath may have a putrid odor although this is not always the case With gradual starvation the patient loses much weight becomes dehydrated cachectic and constipated and shows signs of nutritional deficiencies Sometimes the symptoms of complications may constitute the chief complaint These include hoarseness from recurrent laryngeal involvement cough from aspiration of regurgitated material or from such material entering the bronchi through fistulas hemoptysis anorexia a lump in the neck enlarged abdomen and various bone and muscle pains

### DIAGNOSIS

The history of constant progressively increasing dysphagia is characteristic together with the evidences of starvation The appearance of the regurgitated material not as large in amount and not containing as much mucus as in cardiospasm is also an important feature especially if blood is present If the diagnosis is to be made early enough to make operation possible and successful the public must be instructed to look for help with the first symptoms of slight difficulty in swallowing

### LABORATORY FINDINGS

Persistent interruption of the passage of a large stomach tube or a Rehfuß tube even though the tube is allowed to remain and gentle pressure attempted is an important finding When the tube has met with an obstruction pinching the tube tightly at the level of the teeth drawing it out still pinched and then allowing the contents of the tip of the tube to drop into a white container will usually disclose some blood if a cancer is present A block made of this material will clinch the diagnosis when as is frequently the case particles of cancer tissue are found on microscopic examination A Papanicolaou stain may disclose carcinoma cells I have been able at times to make a differentiation in this way in cases in which some spasm above the carcinoma had confused the x ray and endoscopic observations It takes but a few minutes to make this examination and can do no harm as long as too violent passage of the tube is avoided

### X RAY DIAGNOSIS

A preliminary esophageal aspiration or lavage is of value Fluoroscopy will show obstruction and sometimes the irregular outline of the esophagus

gus at above and below the obstructed area. At times a spasm or a smooth narrowing above the lesion may lead to confusion. Lesions in the mediastinum and lungs may be seen. It is best to take multiple films of the esophagus during the act of swallowing (esophagograms) in different positions to enable a careful study of the details. Dilatation of the esophagus above the obstruction may occur but is never as great as that in advanced cardiospasm (Fig. 28 a). Films taken after the ingestion of only a swallow or two of a thick barium paste or of a capsule filled with bismuth may show a point of narrowing which might be missed after a large barium meal. Films taken at intervals after the full barium meal showing mucosal outlines may disclose a small lesion. In massive involvement both the esophagus and stomach may be seen involved in one mass (Fig. 28 b). In the presence of perforation an esophagobronchial fistula may be disclosed (Fig. 29).

*Esophagoscopy* if it is not interfered with by spasm or smooth narrowing above the cancer will disclose the characteristic nodular appearing and bleeding lesion and a diagnosis can usually be made from its appearance alone. Even a little bloody fluid oozing up through a



Figure 28 a Carcinoma of esophagus showing (1) irregular defect below middle third b Carcinoma of esophagus and stomach showing (1) defect of lower esophagus (2) fundus of stomach with irregular outlines





Figure 29 Esophagobronchial fistula showing (1) site of carcinoma of esophagus (2) and (3) barium entering bronchi (4) bronchial tree containing barium

smooth narrowing is suggestive. A biopsy is safe and should be performed to establish the exact nature of the lesion although of course a specimen taken from a narrow area above the lesion would be of no value. Suction provides material for cytologic examination. What looks like carcinoma may turn out to be a lymphoma of some type, a benign tumor or ulcer, a syphilitic gumma or a mycotic lesion.

A general study of the patient should of course be conducted to determine his status as to possible operative procedures. Cardiovascular, pulmonary, renal and blood studies should be carefully conducted. Evidence of distant metastases would contraindicate any but palliative treatment.

### TREATMENT

As in other parts of the body, there is no cure for esophageal cancer other than extirpation. In inoperable cases, palliative measures may be carried out. Radiotherapy by x-ray, radium or radioactive isotopes in general shows better results with lymphoma and squamous cell carcinoma, but in my experience has not been curative. Although sometimes palliative, the many disagreeable effects of such therapy hardly justify its use. Forceful insertion of more or less rigid tubes through the con-

stricted area (a dangerous procedure) and the old type of gastrostomy for the purpose of feeding contribute but little if at all to the duration of the patient's life and usually increase his suffering. Gastrostomy with insertion of a more or less permanent nylon prosthesis which prevents leakage permits of successful liquid feeding. The feeding of such liquids as the stricture or prosthesis will permit to pass contributes to the patient's comfort while they can still be taken. I do not approve of attempting to prolong life by parenteral feedings but at times glucose and saline solution may make the patient more comfortable by overcoming dehydration. Enemas may be given at long intervals if there is discomfort from distention. Sedation is most humane and toward the end should usually be used to such a degree that the patient does not realize that he is actually dying. Opium and its derivatives are best for this purpose.

### Surgical Treatment

In early cases operation is becoming increasingly successful although only a small proportion of patients survive the five year period metastases or recurrences being prone to occur. Resection of large sections of the esophagus at times together with surrounding tissues and glands is today being done not only for carcinoma of the lower esophagus but even of the cervical esophagus. In the neck anastomoses have been made between the pharynx and esophagus in the chest between the esophagus and the stomach or jejunum which has been so mobilized that it can be brought up and joined to the uninvolved esophagus. Thus far these operations have given some encouragement to the surgeons and some patients have been able to live comfortably after them but on the whole the operative treatment cannot be said to be an unqualified success. With earlier diagnosis and operation there would of course be much better results. Postoperative feedings at first parenteral later oral and general care of complications are essential (see Treatment p 54).

*External pressure* from malignant tumors of the mediastinum lungs and pleura causing obstruction may be complicated by actual involvement of the esophagus in the malignant growth. Dysphagia and regurgitation will accompany the other symptoms of the *primary lesion* and with involvement of the esophagus the symptoms of intrinsic cancer will supervene.

As in other causes of obstruction passage of a stomach tube will be impeded and blood will be obtained if the esophagus has been invaded. X ray studies (Fig 30) will show the defect produced by pressure of the tumor with an irregular outline if the esophageal mucosa has become involved. The treatment will be that of the primary lesion usually with a bad prognosis.



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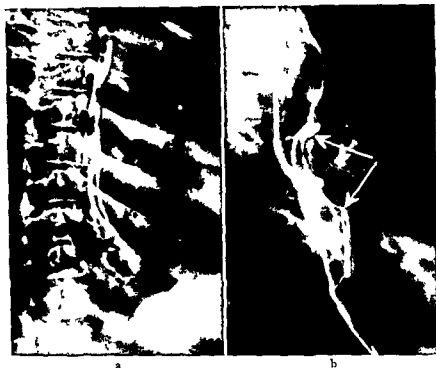


Figure 31 Benign tumors of esophagus *a* Defects due to multiple polyps *b* Defect due to leiomyoma indicated by arrows

X ray films may reveal discrete smooth defects within the lumen or along the wall which will be opaque in solid tumors translucent in cystic tumors (Fig 31) Multiple tumors or those undergoing malignant degeneration may appear irregular

*Esophagoscopy* with biopsy will disclose the nature of the tumor

#### TREATMENT

Benign tumors because they may undergo malignant change should be removed whenever practicable preferably by means of fulguration Some may require surgical extirpation especially if they show signs of malignant degeneration

#### LEUKOPLAKIA

Leukoplakia has been mentioned as rarely occurring in the esophageal mucosa and then usually as an extension from the disease in the mouth It causes substernal distress and perhaps burning during the passage of food through the esophagus with little if any dysphagia The findings would be similar to those seen in esophagitis but the white plaques of leukoplakia can easily be identified on esophagoscopy The development



*Figure 30* Esophageal stricture due to Hodgkin's disease of mediastinal glands. Arrows show extent of pressure on esophagus which is dilated above

#### STENOSIS DUE TO BENIGN NEOPLASMS

Benign tumors rarely occur in the esophagus although all varieties of tumors have been reported polyps being the most frequent. All are slow growing unless malignant changes occur. Polyps may undergo carcinomatous degeneration. leiomyomas may become sarcomatous. Although most benign tumors occur in the upper esophagus multiple polyps may develop at the lower end and may then be confused with varices.

#### SYMPTOMS

Symptoms vary with the size and location of the lesion. A small tumor may present no symptoms or may cause a slight distress when it is pulled down with swallowing. Large tumors may actually obstruct the esophagus and produce the symptoms of stricture. Any of the tumors may bleed with trauma but this is particularly true of multiple polyps of the lower esophagus. Bleeding may also be an indication of malignant degeneration.

#### DIAGNOSIS

The stomach tube may meet with an obstruction may feel as if passing over a bump and may disclose blood on its tip.

## SYPHILIS

Although rarely involving the esophagus syphilis may in the secondary stage cause some dysphagia as a result of mucous patches and in the tertiary stage gummas may exhibit all the symptoms and even the x ray findings of cancer. Careful serologic study and a trial course of anti-luetic therapy will help rule out this disease. Old cicatricial strictures may persist after such therapy (Gastrointestinal Syphilis p 618)

## MYCOTIC DISEASES

The esophagus is rarely invaded by the systemic mycotic diseases (see Mycotic Diseases p 623)

*Actinomyces* occurring in the thorax may involve the esophagus causing obstruction and sinus formation. The symptoms resemble those of cancer but the diagnosis is made certain by the presence of the other involvements.

In patients with moniliasis who show pulmonary or gastrointestinal invasion by *Candida albicans* granulomatous lesions may rarely be seen in the esophagus producing some obstruction with dysphagia. Powdered Mycostatin swallowed with a little water may clear up the infection as it does in the intestine.

*Blastomycosis* by invading lymph nodes in the mediastinum may cause some esophageal obstruction.

*Coccidiomycosis* and *histoplasmosis* with their resemblance to tuberculosis may also involve the esophagus. Mycotic diseases are discussed on page 623.

## VARICES

Varices of the lower esophagus and adjacent stomach occur as a result of portal hypertension mostly when due to hepatic cirrhosis but also in so-called Banti's disease. The former occurs usually in patients in the older age groups the latter mostly in young patients (see chapter on liver disease p 467). The varices vary in size and extent some being small and confined to the lower inch or two of the esophagus some very large usually three in number and extending upward in longitudinal columns for as much as 4 or 5 inches or more. They may cause no interference with the passage of food or may be so large as to cause definite dysphagia. Once formed they usually remain although relief of the portal hypertension may cause some decrease in distention and size and make them less dangerous. The great danger is massive hemorrhage from rupture of a varix formerly considered invariably fatal. Slight bleeding and sometimes even massive hemorrhage may occur from erosion or ulceration of the mucosa over the varices. This constitutes an ulcerative esophagitis over the varices.

of keratosis on the surface of the lesion may cause considerable narrowing and is a recognized precursor of carcinoma. The *treatment* consists in determining and eliminating the cause of the irritation which is usually the swallowing of tobacco juice by chewers or smokers and more rarely of other irritants. X-ray therapy may be advisable when keratosis is developing.

### COLLAGEN DISEASES

*Scleroderma* of the progressive type a rare skin disease of unknown etiology characterized by areas of firm hard skin bound down to the underlying tissues and tending to involve particularly the face, arms and hands, may at times involve the mucosa of the respiratory and gastrointestinal tracts (p. 99). In the latter the esophagus is most frequently affected producing the symptoms and signs of esophagitis with a tendency to the production of stenosis usually of the lower end although there may be multiple areas of narrowing. The esophageal symptoms must be treated as in esophagitis and strictures should be carefully dilated. The disease is usually progressive in spite of any presently known treatment although large doses of vitamins B and C with bioflavonoids, histamines and even sympathectomy have been tried. Recently corticosteroids have produced some remissions at times and may be used to prevent recurrences. Careful observations for complications of this therapy are necessary.

*Other collagen diseases* such as lupus erythematosus, dermatomyositis and polyarteritis nodosa are occasionally accompanied by similar lesions in the esophagus. The whole subject of collagen diseases is discussed in page 99.

### PEMPHIGUS

In patients with typical bullous skin eruptions of pemphigus esophageal symptoms may be due to similar bullae in the esophagus. This disease treated in the past with the same kinds of medication as scleroderma and considered invariably fatal has also recently been treated with some success by means of steroid therapy.

### TUBERCULOSIS

In advanced pulmonary tuberculosis the larynx and contiguous hypopharynx are frequently involved producing dysphagia. Although this dysphagia may occur without actual involvement of the esophagus obstruction of the esophagus may be the result of pressure from mediastinal lymph node involvement. Cicatricial contractions of such areas by pulling on the esophagus cause traction diverticula. These may rupture into the left bronchus. Fortunately such severe lesions are rarely encountered with the newer methods of therapy (p. 615).

performed in order to prevent rupture of the varices. The careful *passage of a soft rubber tube* or Richfuss tube will do no more harm than food and may disclose a slight delay in passage through the lower esophagus and possibly a little bleeding from surface erosion. No attempts at forcing it through should be made and the tube should be promptly removed.

A *ray study* done carefully will almost invariably show distortion and an irregular outline of the lumen sometimes giving the exact outline of the varices (Fig 32). This examination should be done at various angles with barium mixtures of different consistencies and with the patient standing and lying down. As varices are frequently overlooked on fluoroscopy, multiple films should always be made. In some cases multiple polyps or even cancer may be mistaken for varices.

*Esophagoscopy* should be performed to rule out tumors, great caution being exercised to prevent injury. Careful gastrointestinal study should be done to rule out other sources of bleeding, especially if no bleeding is seen in the esophagus.

#### TREATMENT

Varices which are small and have no history of bleeding or obstruction usually require no local treatment but the liver and spleen should receive careful attention medically or surgically (See chapter on liver disease p 467). Ruptured bleeding varices require prompt attention before the patient bleeds to death. The first step should be elevation of the foot of the bed 10 inches or more which will frequently stop the bleeding. Transfusion may be necessary at once although it is better to postpone this until the bleeding has been stopped. Every emergency clinic and ambulance should be equipped with a gastric balloon, a stomach tube with an inflatable balloon at its tip for so-called cardio esophageal tamponade. It was formerly considered that a double balloon arrangement was necessary so that when the tube was passed into the stomach the inflated lower part of the balloon would keep the tip of the tube in the stomach and the upper one would compress the varices throughout their length. It has since been found that as the veins run upward merely pulling one balloon firmly up into the cardia will stop most hemorrhages promptly from both esophagus and stomach so that the patient can safely be given transfusions and parenteral feedings. However removal of the balloon even days later will often bring on an immediate recurrence of hemorrhage or the bleeding may frequently recur later. It is most desirable to stop the bleeding by actually placing sutures around all the varicose veins through a trans thoracic approach and opening of the fundus of the stomach and lower esophagus so that the veins and even the bleeding point can be visualized. This operation should be performed as soon as the bleeding has



## SYMPTOMS

Varices may produce no symptoms the patient merely having the general symptoms of the condition causing the portal hypertension. When the varices are large enough to cause interference with swallowing more or less dysphagia may be present and regurgitation may occur as in any case of stenosis. Pain is rarely present and is then the result of mucosal erosion or ulcer. When massive hemorrhage takes place it may be so extensive that more than half of the patients will die within a short time unless treatment is started at once. Even when the bleeding has stopped unless definitive treatment of the cause of the varices is carried out successive hemorrhages will threaten the life of the patient.

## DIAGNOSIS

Even in the absence of esophageal symptoms a patient with hepatic cirrhosis or Bantus syndrome should be carefully studied to rule out varices. Patients who have had unexplained hematemesis also require this investigation. It must be realized however that over 25 per cent of hemorrhages in patients with cirrhosis come from gastric or duodenal ulcers.

Examination of the esophagus by the usual methods must be gently



Figure 32 Esophageal varices. Note discrete and confluent defects of lower esophagus and adjacent stomach.

and with general symptoms of vitamin B complex deficiency is an entity easily distinguishable as a rule from these somewhat similar conditions.

Passage of a stomach tube may not be impeded and is used for gastric analysis. The x ray film may show nothing abnormal or may disclose slight slowing of the barium meal in its passage through the esophagus. Esophagoscopy will frequently show the mucosal changes resembling those seen in the mouth and pharynx.

*Similar symptoms may occur in cancer, pernicious anemia and other diseases elsewhere and all other possible causes should be ruled out by a complete gastrointestinal survey.*

### TREATMENT

Passage of a stomach tube as a therapeutic measure but even when used merely for the study of gastric secretions will usually relieve the dysphagia sometimes permanently. A well balanced nutritious diet with the addition of ferrous salts and mixed vitamins and with special attention to the B complex will usually overcome the deficiencies and the patient should be admonished to follow a normal diet with added vitamins and minerals for an indefinite period. Blood transfusions are rarely required and medication should be avoided.

### FOREIGN BODIES

All kinds of foreign bodies have been intentionally or accidentally swallowed not only by children but also by adults. It is natural for children or the insane to put all kinds of articles into their mouths and in some occupations tacks, nails and pins are held in the mouth during work. Swallowing them is not uncommon. In most cases even surprisingly large and sharp articles will pass through the gastrointestinal tract without causing symptoms. Trouble comes when the articles stick at some point in the esophagus usually at one of the naturally narrow areas at the cardiac orifice or at a previously unsuspected lesion producing moderate stenosis. When the foreign body becomes impacted it may produce more or less complete obstruction or if it is pointed or has a sharp edge it may perforate the esophagus into adjacent thoracic structures or the lesser peritoneal cavity.

### SYMPTOMS

In the absence of a history of the swallowing of a foreign body dysphagia may be the principal symptom or severe pain without noticeable dysphagia may occur. Bleeding may at times be profuse then producing both hematemesis and melena. Perforation will produce the symptoms of perforation from other causes those of an intrathoracic or intra-abdominal calamity.

been stopped by the tamponade and should be accompanied by transfusion. The mortality of this procedure should not be over 10 per cent in expert hands.

Even though these combined methods will save life temporarily it has been found that with no reduction in the portal hypertension recurrent bleeding can usually be expected. Therefore a suitable shunting operation such as portacaval shunt should be performed within a few weeks except in patients whose liver damage is so extreme or whose prothrombin deficiency, deficiency of serum globulin, high bromsulphalein retention, hyperbilirubinemia or ascites is so persistent that such an operation is useless and contraindicated. In Bant's syndrome splenectomy combined with the shunting operation is the most satisfactory procedure (splenorenal shunt).

#### PLUMMER VINSON SYNDROME

First described by Plummer in 1914 and elaborated upon in 1922 by Vinson, the Plummer Vinson syndrome is a condition in which dysphagia associated with iron and vitamin deficiency are the cardinal symptoms. There is still some difference of opinion whether dysphagia comes first followed by the nutritional deficiency because of inability to swallow food or whether the disease is primarily a hypochromic microcytic anemia usually associated with achlorhydria or hypochlorhydria. There is usually a glazing or atrophy of the mucosa of the tongue, mouth, pharynx, esophagus and stomach, perhaps as a result of vitamin B complex deficiency. Occasionally splenomegaly has also been noted. Narrowing due to the mucosal changes may be comparatively slight but may stiffen the esophageal mucosa enough to produce difficulty in swallowing. The term hysterical dysphagia has been used to describe this condition but neurotic symptoms are undoubtedly its result rather than its cause. The fact that it occurs mostly in women near the menopause suggests a possible endocrine etiology. This syndrome must be differentiated from several somewhat similar conditions.

#### DIAGNOSIS

Neurotic patients are frequently seen who say they "cannot swallow food." In some cases of anorexia nervosa patients ward off feedings by this complaint. Some merely use this term as an excuse for not eating because they have symptoms somewhere in the abdomen after eating. Such cases will be discovered on careful gastrointestinal study. *Globus hystericus*, a condition in which the patient imagines a lump in the throat or upper esophagus but can swallow if urged, occurs usually as a result of some severe nervous upset. The Plummer Vinson syndrome occurring in women near the menopause consisting of dysphagia associated with the blood, mucosal and secretory changes mentioned above

and with general symptoms of vitamin B complex deficiency is an entity easily distinguishable as a rule from these somewhat similar conditions.

Passage of a stomach tube may not be impeded and is used for gastric analysis. The x ray film may show nothing, abnormal or may disclose slight slowing of the barium meal in its passage through the esophagus. Esophagoscopy will frequently show the mucosal changes resembling those seen in the mouth and pharynx.

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## DIAGNOSIS

With a history of ingestion of a foreign body it is best not to attempt the passage of a stomach tube since blind pressure against or dislodgment of a perforating body might produce grave consequences. X ray films will disclose the location of radiopaque substances such as pins ticks nails dental plates coins metal buttons fasteners and the like. Nonopaque articles such as fish or small chicken bones food bolus glass plastic buttons parasites (*ascaris*) and methyl cellulose capsules will rarely be seen even with the swallowing of barium to outline them although a spasm or narrowing will suggest their presence. Swallowing a small pledget of cotton impregnated with barium or bismuth may cause it to be caught on a bone and disclose its presence.

## TREATMENT

*Esophagoscopy* will not only locate the foreign body but also will provide the method of removing it in most cases special instruments having been devised to facilitate the removal of various articles. After their removal the patient should be observed carefully for possible perforation which would require operation. If the impacted article was small indicating that the lumen was narrowed a thorough esophageal study to determine the cause of narrowing should be instituted. Occasionally it is necessary to remove the foreign body through an incision into the esophagus.

## RUPTURE OF THE ESOPHAGUS

The esophagus may be ruptured as a result of (1) disease as in perforation of an ulcer or cancer or of trauma (2) instrumentation or foreign bodies or pneumatic pressure (3) puncture wounds or crushing blows or falls (4) rupture of an aneurysm or abscess into the esophageal lumen (5) It may also rupture spontaneously the rupture being usually vertical and located not far above the cardiac orifice. Spontaneous rupture occurs mostly in chronic alcoholics and usually follows severe vomiting from any cause after alcohol and a large meal or after severe retching when trying to bring up food impacted at an obstructing lesion.

## SYMPTOMS

Esophageal rupture from any cause presents certain characteristic symptoms. Sudden agonizing pain in the chest particularly in the sternal region and in the abdomen especially in the epigastric region with a history of the causes mentioned above is the first symptom. In the case of a ruptured aneurysm profuse hemorrhage with prompt exitus is the rule and rupture of an abscess will be followed by regurgitation of pus. When the pleura has been punctured pneumothorax occurs quickly. Shock is an early symptom. If the true nature of the condition is not

suspected and nothing is done for it there will follow evidences of a gross infection of the mediastinum pleura lung pericardium and peritoneum with their usual severe symptoms and sequelae. The most spectacular finding when the mediastinum is involved is the infiltration of air under the skin (emphysema) spreading to neck face thorax abdomen and even extremities with crepitation on examination.

#### DIAGNOSIS

With trauma the history will usually suggest the diagnosis although acute alcoholism may obscure the symptoms. X ray films taken early will show the presence of air in the mediastinum pleura pericardium or peritoneum later they will reveal fluid in these locations. Aspiration of the fluid will often show contamination with food and at times hydrochloric acid and cultures may disclose gastrointestinal flora. The patient may be able to swallow and if in error barium or other radiopaque substance has been administered it will be seen on x ray outside the lumen of the esophagus. Instrumentation must be avoided.

#### TREATMENT

Operation offers the only hope of saving life. It should be performed at once when the classic symptoms occur transfusions antibiotics and supportive measures being used as part of the preparation for the operation and during the operation. A rent may be merely closed up or a partial resection may be necessary. Gastrostomy may be done for temporary feedings while the esophagus is healing or liquid feedings through a fine polyethylene tube may be given. Adequate treatment of the complications must be instituted and attention given to proper nutrition.

With gross contamination of surrounding structures even lavage through the operative wound drainage and massive doses of antibiotics will often not prevent a fatal termination.

## DIAGNOSIS

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## TREATMENT

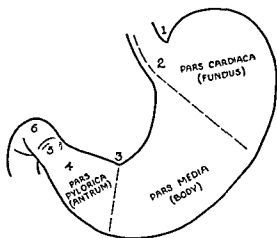
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- 1 *Incisura Cardiac*
- 2 *Cardia*
- 3 *Incisura Angularis*
- 4 *Pyloric Canal*
- 5 *Pylorus*
- 6 *Duodenal Cap*

Figure 33 Anatomic divisions of the stomach (Boxkus Gastroenterology Vol I)

or regional lymph nodes may push the stomach upward and forward. The thick apron of the gastrocolic omentum is a protection from injury. Tumors of any of these locations may actually invade the stomach wall.

The stomach wall has four layers. The outer peritoneal or serous coat, a reflection of the peritoneum, is thin but very tough and elastic, providing protection from external injury. The muscular coat consists of powerful muscle fibers running both transversely and longitudinally, which are more distensible in the fundus. The submucosa varies in thickness and consists of loose areolar tissue through which run vessels and nerves. It also contains its own muscular coat, the muscularis mucosae. The mucosa, the inner lining of the stomach, is very tough and resistant to injury. It is thrown into folds which smooth out when the stomach becomes distended. It contains various secreting cells. The cap contains Brunner's glands. The mucosa has remarkable healing qualities; injuries, excisions, burns, and even ulcers heal rapidly. The resulting deformities vary from shallow and hardly noticeable scars to large deforming areas of scar tissue.

The blood supply is rich and is mainly from branches of the celiac axis. In most of the stomach the vessels supplying the mucosa anastomose freely with each other, assuring an adequate supply of blood to the important secreting cells. However, along the lesser curvature, the im-



# The Stomach

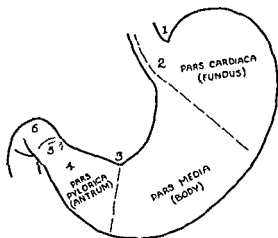
## General Discussion

In spite of the fact that gastrectomy has become one of our most frequent and approved operations it is still true that the human stomach is of great importance not only because of its digestive function but also because of its contribution to general health and happiness. Ask the person who has had the whole or even a part of his stomach removed Strange to relate although research in regard to the stomach has been carried on constantly and intensively especially since Beaumont's epoch making discoveries there is yet no unanimity of opinion about even the anatomy and physiology of this organ. It is therefore not surprising that so much difference of opinion exists about the nature and treatment of its diseases. As this is a book on clinical gastroenterology it cannot cover the whole field of anatomy and physiology of the stomach. I shall therefore confine myself to a presentation of some of the important facts having a bearing on the clinical approach to diseases of the stomach.

## ANATOMY

The stomach consists of three main divisions to each of which different names have been given. The first portion a distensible reservoir situated largely above the level at which the esophageal contents enter it through the cardia is called the *fundus* the fornix or just the cardiac end. The midportion called the *corpus* the body or the pars media extends to the *incisura angularis* beyond which is the *antrum* or pyloric end of the stomach. The pylorus is opened and closed by a sphincter or valve according to different authors. Beyond this lies the *cap* also called the duodenal bulb or first portion of the duodenum. It was named the cap by Lewis Gregory Cole who pointed out that anatomically physiologically and embryologically it belongs more to the stomach than to the duodenum.

The stomach has anterior and posterior walls and a lesser curvature and a greater curvature. It lies close to or in contact with other organs enlargements of any one of which may alter the position of the stomach as follows: (1) An enlarged liver and spleen may push it to right or to left. (2) An enlarged gallbladder may compress the lesser curvature of the antrum and cap. (3) An enlarged pancreas may compress its posterior wall and its greater curvature and may narrow the whole lumen of the cap and duodenum. (4) An enlarged transverse colon may compress the greater curvature. (5) Retroperitoneal enlargements as of the kidneys



- 1 *Incisura Cardiaca*
- 2 *Cardia*
- 3 *Incisura Angularis*
- 4 *Pyloric Canal*
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- 6 *Duodenal Cap*

Figure 33 Anatomic divisions of the stomach (Bockus Gastroenterology Vol 1)

or regional lymph nodes may push the stomach upward and forward. The thick apron of the gastrocolic omentum is a protection from injury. Tumors of any of these locations may actually invade the stomach wall.

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The blood supply is rich and is mainly from branches of the celiac axis. In most of the stomach the vessels supplying the mucosa anastomose freely with each other, assuring an adequate supply of blood to the important secreting cells. However, along the lesser curvature the im-

mediate prepyloric region and the anterior and posterior surfaces of the cap the mucosa is tightly bound to the submucosa and the blood is supplied by end arteries which have no anastomoses. Any stasis or narrowing of these end arteries will therefore cut off the blood from the area supplied resulting in necrosis and surface sloughing—in other words an ulcer.

The *nerve supply* is from both the sympathetic and parasympathetic (vagus) systems.

### PHYSIOLOGY

#### Automatic Control of Gastric Functions

The obvious function of the stomach is the digestion or partial digestion of ingested food to prepare it for complete digestion and absorption by the *small intestine*. This is accomplished by the *gastric juice* consisting essentially of pepsin, hydrochloric acid and mucus although other agents including at times a hyperglycemic factor (amylase), the antianemic factor and several hormones are also secreted by the gastric mucosa.

*Hydrochloric acid* is secreted by the parietal cells of the mucosa mostly of the corpus and fundus. Its secretion is stimulated by the hormone gastrin, probably identical with histamine. This is produced by contact of food and other chemicals with the mucosa of the antrum by external hormones such as insulin and by vagal stimulation. *Pepsin* is fairly continuously secreted by the surface epithelium and the chief cells near the cardia. *Mucus* is secreted continuously throughout the stomach. It protects the gastric mucosa from irritation by stomach content and from digestion or erosion by the acid; its concentration and quantity varying according to the need for such protection. Even in the absence of mucus, however, the normal living gastric mucosa cannot be digested by the gastric juice as has been shown by suspending fragments of gastric mucosa in the stomach lumen through fistulas. This fact must be remembered in discussing the etiology of ulcer. The mucus is secreted constantly profusely and persistently, influenced only in the direction of overproduction in the presence of irritation. Pepsin is also fairly constantly secreted, being found in the gastric contents even in the absence of hydrochloric acid. A milk curdling ferment, *rennin*, was formerly thought to be present.

The variations in acidity occurring in the stomach secretion have never been fully explained. Nor has it ever been definitely established whether the individual parietal cells produce varying amounts of hydrochloric acid according to the degree of stimulation or the amount of food remaining in the stomach, whether regurgitation of alkaline duodenal content accounts for the decline in acidity at the end of a meal or whether the persistent hypersecretion present in certain diseases such as peptic

ulcer is due to an actual increase in the number of parietal cells. One thing however has been definitely established namely that a high degree of acidity does not in itself occasion any symptoms and is not the cause of pain in peptic ulcer.

*Heat regulation* is another important function. Food reaching the stomach at low temperatures is rapidly raised to body temperature or a little higher by producing an extra flow of blood through the many blood vessels. Foods at high temperatures are less efficiently cooled to body temperature because of the smaller differential in the temperature between the hot content and the blood. Excessively hot foods like coffee and soups therefore will lie in the stomach at a high temperature long enough to cause real irritation almost burning the mucosa. A history of taking such hot liquids habitually is frequently obtained in patients with gastric cancer.

The *antiseptic action* of the gastric juice is also important. Most ordinary swallowed live organisms are destroyed by digestion although of course there are parasites ova and some bacteria and viruses which can survive and become active in the intestine.

*Absorption* of water and aqueous and alcoholic solutions has been shown to occur although at a much slower rate than absorption from the small bowel. In experiments with heavy water it has been estimated that 25 per cent per minute is the rate of absorption from the stomach. In allergy experiments definite distant reactions have occurred within four minutes of ingestion of the allergen.

*Digestion* in the normal stomach is carried on in an orderly but somewhat flexible manner being influenced by a variety of internal and external factors as follows

1 The *consistency* of the material ingested. *Liquids* pass rapidly through the stomach into the duodenum *cold liquid* at times more rapidly than warm. A glass of cold water may leave the stomach in fifteen or twenty minutes. The emptying time is progressively slower for foods of greater consistency some solid foods remaining in the normal stomach as long as three to five hours. Liquids consumed with solids or produced by the digestion of solids are expelled even though the solids remain. Water taken with a meal is thus prevented from diluting the gastric juice and interfering with digestion.

2 The *pH* of the ingested food or other material influences the secretion of hydrochloric acid. Because an acid medium is necessary for peptic digestion the ingestion of soluble alkalis such as sodium bicarbonate produces the greatest outpouring of hydrochloric acid. On the other hand ingestion of mineral acid inhibits the secretion of hydrochloric acid.

3 The *character* of the food ingested has a definite effect on both secretion and motility. Any kind of food put into the stomach will initiate the secretion of gastric juice. Even water and saline solution

although they remain but a short time produce a definite secretion. Saliva is a natural stimulant. The greatest stimulants to secretion are meat and meat extractives and peptone. Carbohydrates even glucose are milder stimulants. Fats although producing some secretion also stimulate the production of enterogastrone which inhibits secretion and motility. Organic acids lactic butyric and acetic acids and the fruit acids are definite stimulants. Alcohol in moderate amount is an excellent stimulant to secretion but inhibits secretion when taken to excess. Regurgitation of bile and pancreatic juice which are alkaline in reaction stimulates secretion to some extent. Alumina and silicates do not stimulate secretion. The alkali sulfites stimulate an outflow of mucus.

4 *Hormones* stimulating gastric secretion are gastrin or histamine which is supposed to be secreted by the stomach and epinephrine reaching the stomach through the blood stream. Insulin stimulates acid secretion hypoglycemia causing marked hyperacidity and hypersecretion. Enterogastrone and urogastrone inhibit secretion.

5 *Vitamins* Vitamin B complex has been considered a stimulant to gastric secretion but this has not been proved. Vitamin D by increasing blood calcium causes a diminution in acidity.

6 *The condition of the stomach influences both secretion and motility.* When the stomach is weakened from excessive general fatigue or from debilitating disease elsewhere secretion and motility are diminished. Diseases of the stomach itself or pressure from without may be accompanied by an increase or a decrease in both secretion and motility. The effects of disease will be discussed later.

The functions just discussed motility and secretion are *automatic*. They have been shown to take place in stomachs completely cut off from external nervous influences and in human stomachs into which food was passed through a tube. Solid foods will be held in the fundus at first permitting salivary starch digestion to go on for a while. When the acid gastric juice thoroughly mixed with the contents by muscular contractions has produced an acid medium the ptyalin of the saliva is inactivated and peptic digestion is initiated. As the contents become softened or chymified they go through a more strenuous crushing action in the corpus and then in the antrum. Finally the powerful fan shaped muscle of the distal antrum first described by Cole squeezes the contents like a lemon squeezer in a mass contraction against the pylorus which permits liquid or very thin contents to go into the cap where they rest for a short time before going further into the small intestine there to be fully digested and absorbed. After this mass contraction of the stomach there is a period of relaxation when the contents of the entire stomach are mixed together. The process is then repeated. Cole demonstrated that peristalsis in the stomach producing the propulsion of contents is carried on by the muscularis mucosae. The more powerful muscular

coat holds the contents firmly by mere tonus and mixes them with its contractions

At the end of the digestive period substances which are completely indigestible and unable to be reduced to a liquid or even a soft consistency may be pushed through the pylorus which has become relaxed for the purpose. If too large to be vomited they may lie in the stomach for long periods of time. Table silverware in quantity has frequently been removed from the stomachs of mental patients by repeated operations at intervals of a year or more. When the stomach is finally empty or nearly so there is a varying period of resting during which there is a continuous but minimal amount of secretion of hydrochloric acid and pepsin and continuous secretion of mucus. The muscles also rest for a time although a moderate degree of tonus is maintained.

After a period varying from one to three or more hours the tonic contractions become more powerful often almost tetanic and increasingly forceful. They are then called *hunger contractions*. The person senses these contractions as hunger pangs. At first they occur intermittently but later become continuous being relieved at once by ingestion of food or other material. In prolonged fasting these contractions gradually disappear and all hunger sensation is finally lost. As a result of irritation from injury or disease as in the presence of ulceration the hunger contractions become so powerful and persistent that they are felt as a pain which as in the case of hunger pangs may be more or less intermittent and relieved by food.

### Nervous Control of Gastric Functions

So far only the automatic physiology of the stomach has been discussed and I have attempted to make clear that the stomach will do its work independently of nerve control. This fact is too frequently overlooked by both clinicians and surgeons who have become psychosomatic minded and treat all gastric conditions through attention to the nerves. There is however a definite nerve control of gastric function which supplements the automatic function at times aiding at other times inhibiting it.

There are three types of nerves distributed in the stomach wall. The *sympathetic nerves* originating in the celiac plexus and the parasympathetic from the right and left *vagus nerves* run separately or together and activate muscles and glands. In addition to these there are *intramural plexuses* of nerves the *myenteric* or *Auerbach's plexus* situated between the fibers of the longitudinal muscles the *submucosal* or *Meissner's plexus* distributed to the submucosa and the *subserous plexus* located below the serous coat. The exact functions of each of these types of nerves in the stomach is still very much in dispute by physiologists. It is felt that the plexuses guide the intrinsic automatic activities of the

although they remain but a short time produce a definite secretion. Saliva is a natural stimulant. The greatest stimulants to secretion are meat and meat extractives and peptone. Carbohydrates even glucose are milder stimulants. Fats although producing some secretion also stimulate the production of enterogastrone which inhibits secretion and motility. Organic acids lactic butyric and acetic acids and the fruit acids are definite stimulants. Alcohol in moderate amount is an excellent stimulant to secretion but inhibits secretion when taken to excess. The regurgitation of bile and pancreatic juice which are alkaline in reaction stimulates secretion to some extent. Alumina and silicates do not stimulate secretion. The alkyl sulfates stimulate an outflow of mucus.

4 *Hormones* stimulating gastric secretion are gastrin or histamine which is supposed to be secreted by the stomach and epinephrine reaching the stomach through the blood stream. Insulin stimulates acid secretion hypoglycemia causing marked hyperacidity and hypersecretion. Enterogastrone and urogastrone inhibit secretion.

5 *Vitamins* Vitamin B complex has been considered a stimulant to gastric secretion but this has not been proved. Vitamin D by increasing blood calcium causes a diminution in acidity.

6 *The condition of the stomach* influences both secretion and motility. When the stomach is weakened from excessive general fatigue or from debilitating disease elsewhere secretion and motility are diminished. Diseases of the stomach itself or pressure from without may be accompanied by an increase or a decrease in both secretion and motility. The effects of disease will be discussed later.

The functions just discussed motility and secretion are *automatic*. They have been shown to take place in stomachs completely cut off from external nervous influences and in human stomachs into which food was passed through a tube. Solid foods will be held in the fundus at first permitting salivary starch digestion to go on for a while. When the acid gastric juice thoroughly mixed with the contents by muscular contractions has produced an acid medium the ptyalin of the saliva is inactivated and peptic digestion is initiated. As the contents become softened or chymified they go through a more strenuous crushing action in the corpus and then in the antrum. Finally the powerful fan shaped muscle of the distal antrum first described by Cole squeezes the contents like a lemon squeezer in a mass contraction against the pylorus which permits liquid or very thin contents to go into the cap where they rest for a short time before going further into the small intestine there to be fully digested and absorbed. After this mass contraction of the stomach there is a period of relaxation when the contents of the entire stomach are mixed together. The process is then repeated. Cole demonstrated that peristalsis in the stomach producing the propulsion of contents is carried on by the muscularis mucosae. The more powerful muscular

and often definite hypermotility. Neoplasms usually become eroded and cause bleeding. Deeply penetrating lesions with perforation may result in acute peritonitis or may become walled off by adhesions to neighboring organs such as the pancreas or liver or may be covered by adhesions to the omentum or may invade neighboring organs such as the gallbladder and colon producing fistulas. Local manifestations of general infections may be found such as syphilitic or tuberculous lesions or acute virus infections.

### DIAGNOSIS

As in other parts of the gastrointestinal tract the diagnosis of diseases of the stomach depends upon a careful history, a thorough physical examination and the use of suitable laboratory methods. The three most essential legs upon which a gastric diagnosis must rest are the history, the fractional gastric analysis and the x-ray findings.

#### Symptomatology

In general the principal symptoms produced by gastric diseases are essentially pain and retrostaltic symptoms.

Pain has been discussed under Physiology and the significance of the three kinds of pain has been outlined. The location of the pains although mostly in the epigastrium may at times be entirely substernal or may be referred to or actually located in either side or in the spine. In the case of substernal pains differentiation from cardiac pains is at times difficult especially when both gastric and cardiac lesions are present.

*Retrostaltic or Reflex Gastric Symptoms.* These include the following:

1. Mere *peristaltic unrest* often referred to as "indigestion," fullness, "butterflies" or "gas."

2. Actual *reverse peristalsis* pushing gastric content into the lower esophagus causing heartburn, pushing it up to the pharynx causing sour eructations or to the mouth causing regurgitation usually sour tasting. If sufficiently powerful it will actually cause vomiting. This type of *reflex vomiting* comes immediately after meals the vomitus containing only recently ingested food. It must be differentiated from the *delayed vomiting* of foods eaten a long time before which is caused by obstruction of the pylorus or duodenum. Both types differ from the *projectile vomiting* resulting from a brain lesion involving the vomiting center.

3. Relief of retrostaltic symptoms by *belching* or *aerophagia*. This consists in first closing the lips firmly, swallowing the air in the mouth then bringing it up. The air may be swallowed only as far as the pharynx or upper esophagus and be brought up immediately producing a series of more or less noisy belches or may be swallowed into the stomach often distending the lower esophagus and stomach sufficiently to cause distress or pain. The prompt relief following the loud expulsion of the air gives



stomach while the sympathetic and vagus nerves regulate secretion and motility from without. Impulses from the vagus nerve stimulate both motor and secretory activity in the stomach; those from the sympathetic nerves tend to diminish or depress these functions. A careful balance is therefore necessary. To vagal activity is attributed the first nervous appetite or cephalic phase giving rise to gastric secretion as a result of a person's thinking about food, seeing, tasting or smelling it. To accentuated vagal activity has been ascribed the hyperacidity or hypersecretion and the hypermotility, hypertonicity and spasms associated with disease as in peptic ulcer. It is supposed that insulin stimulates gastric secretion through the vagus nerve. Insulin has therefore been used to determine whether the operation of vagotomy or vagus section has successfully removed all vagal fibers. Such procedures ignore the second chemical or hormonal phase of gastric secretion described before as being automatic and accounting for fully half of all gastric secretion. In diabetes there is often gastric atony.

### Gastric Pain

The stomach is not supplied with sensory fibers so that pain in the usual sense does not occur. There are however certain conditions which cause pain. Excessive hunger contractions have already been mentioned as causing hunger pain as in ulcer. Excessive contraction or spasm at a sphincter such as the cardia and pylorus often reflex or retrostaltic in origin will produce a severe spasmodic pain usually soon after eating. This may also occur in ulcer close to the pylorus. Peritoneal pain moderate in degree may result from excessive stretching of the peritoneal coat by marked distention with food or gas (air) especially from distention of a large ulcer crater in the stomach or cecum. Severe agonizing pain occurs when the peritoneum is punctured or torn as in perforation of an ulcer. Even a stoic will scream with terror from such a pain. Lower esophageal pain or heartburn has been described in the section on the esophagus as being due to distention of the esophageal wall either by swallowed material or regurgitation of gastric content. Acidity does not play an essential role in causing this burning. It may occur when the regurgitated gastric content contains no acid.

### PATHOLOGY

The stomach is the seat of various pathological processes which will be discussed under each specific disease. In general those tending merely to irritate the stomach will be accompanied by hyperactive secretion and peristalsis. Prolonged irritation will produce thickening of the stomach wall with hypertrophy of the mucosa. The processes tending to destroy or injure large areas of secreting mucosa or diseased conditions in the stomach or elsewhere causing its atrophy will show decreased secretion.

that it is present in most cases of ulcer occasionally in cancer more rarely in other conditions. It may be due to many other abdominal conditions however reflected or referred from any part of the gastrointestinal tract or by renal pelvic or even spinal lesions. *Rebound tenderness* present in perforation of a gastric lesion may also occur in peritonitis due to other causes. *Muscular spasm or rigidity* may or may not occur with threatened or actual perforation and a normal relaxed state of the musculature may at times be found in serious stomach lesions. It is therefore important to realize that though physical examination may at times be a big help in diagnosis it may often be deceptive. Physical examination should usually be supplemented by special studies instrumental and laboratory.

**Laboratory Examinations ROUTINE EXAMINATIONS** Some routine examinations are usually indicated. Blood cell counts cultures chemistries sedimentation rate hematocrit serology and other procedures will disclose anemias the degree of concentration evidences of infection the electrolyte balance and the presence of other diseased conditions which might affect the stomach. Urine examinations are important for the same reason. *Stool* examinations are important in suspected hemorrhage positive occult blood tests being used to confirm the presence of blood in questionable black or tarry stools. It must be borne in mind however that the finding of occult blood in the absence of visible blood may be due to slight bleeding from any part of the alimentary canal from the teeth down and from admixture of blood from the respiratory system or the female pelvis. The finding of *undigested meat fibers* and occasionally of undigested starch granules may be due to failure of digestion of the protein envelope or covering of these foods which require peptic digestion.

**SPECIAL EXAMINATIONS** *Gastric analysis* was used before the x rays were discovered and still gives valuable information about the stomach. The most important procedures included under this term are the examination of the fasting gastric content (overnight residue after feeding of half-cooked rice and raw raisins at bedtime) and of specimens removed by means of a Rehfuess tube at fifteen minute intervals after intramuscular injection of histamine. The old method of removing a single specimen of gastric content after a test meal was never of any definite diagnostic value.

The *overnight residue* normally consists of 25 to 75 cc. of gastric juice mixed with gastric esophageal oral and upper respiratory mucus and occasionally with an admixture of bile from regurgitated duodenal contents. Blood may be found from trauma in passing the tube. In *pyloric or duodenal obstruction* the residue may be large or small in amount depending on the degree of obstruction since liquid will pass through a lumen not permitting the passage of solids. Large amounts allowed to

the impression that the distress was caused by gas. It prompts the patient to seek relief from any kind of distress by inducing belching not only by air swallowing but also by the ingestion of alkalis. The latter combined with hydrochloric acid in the stomach will produce carbon dioxide the belching of which produces such pleasure. If not belched the air or gas may go through the pylorus and cause distention of the small and even the large intestine.

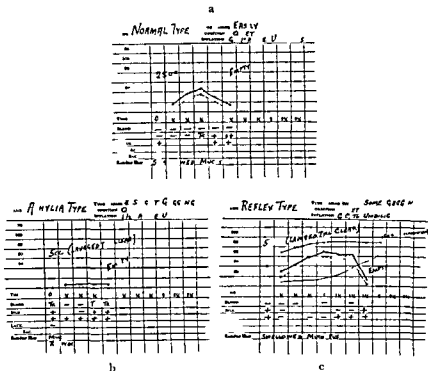
*Nervous and Mental Symptoms* With any kind of gastric symptoms whether acute or chronic the patient becomes disturbed mentally. Fear especially of cancer is common as is fear to eat because it might do harm. Many patients starve themselves needlessly. Anorexia or disgust for food may also cause actual starvation. The patient becomes irritable excitable disagreeable the typical chronic dyspeptic of comic stories and cartoons. It is because of such symptoms caused by stomach troubles that the psychiatrists have built up their explanation of the symptoms as due to mental disturbances. However when the gastric condition is adequately treated medically the mental symptoms clear up rapidly.

*Hemorrhage* Hemorrhage from the stomach may be manifested by the vomiting of blood either bright red clotted or partly digested the "coffee ground vomit." On the other hand even large hemorrhages may cause no vomiting manifesting themselves only by symptoms of internal hemorrhage or shock and by the passage of tarry stools evidence of bleeding from high up. Differentiation from nasopharyngeal or pulmonary hemorrhage is at times difficult. The history may disclose the origin. Blood tinged foam when brought up points to a pulmonary origin.

## Examinations

*Physical Examination* In any case it is always imperative to make a complete examination of every patient. We must realize that disease of almost any organ of the body may affect the stomach that focal infections may cause serious damage and that cardiovascular respiratory renal or pelvic disease may affect the stomach directly or indirectly.

**ABDOMINAL EXAMINATION** On inspection the stomach may be seen distended with swallowed air or gas and may in cases of pyloric stenosis be filled with food. Even when food is not seen a stomach containing some liquid may be somewhat outlined by percussion and the position of the greater curvature may be located by succussion gurgling being induced by a deep push with the fingers. Palpation may locate a mass in the epigastric region. Its relation to the stomach may be determined by distending the stomach with air through a stomach tube or by feeding an effervescent mixture such as sodium bicarbonate and citric acid. In a case of suspected ulcer this should be avoided since a perforation might be induced. The x ray study is better anyway. Tenderness in the epigastrium should not be accepted as evidence of gastric disease. It is true



**Figure 34** Fractional gastric analysis. *a* Normal type. Secretion acid curve down after 1 hour. Motility slight fasting residue empty in 1½ hours. Admixtures bile regurgitated at end, no blood. *b* Achylia type. Secretion no free hydrochloric acid. Motility small overnight residue rapid emptying. Admixtures early bile regurgitation occult blood from congestion. *c* Reflex continued secretion type. Secretion acid curve rising or level till empty. Motility moderate fasting residue rapid or delayed emptying. Admixtures Bile present or not present, no blood.

each fifteen minute interval 5 to 8 cc of content are removed and at the end of the examination the entire remaining content is aspirated and measured. A normal stomach may contain 50 to 75 cc of liquid at ninety minutes and should be empty in two hours. Emptying in one hour or less indicates a hypermotility such as is seen in achylia. A large residue in two hours 100 cc or more is found in an atonic stomach in obstruction and more rarely in simple hypersecretion. A large two hour residue in a patient who had an overnight residue of rice and raisins indicates a small lumen at the point of narrowing. A small or absent two hour residue in such a patient would indicate an obstruction only to solids the size of the rice and raisin particles. A large two hour residue in a patient with no trace of overnight residue would indicate a temporary insufficiency, a pylorospasm or atony or both.

3 *Admixtures* are often important Mucopus may indicate a severe respiratory infection Bile normally occurring toward the end of the

stand in a beaker will usually form three layers with solid material at the bottom light foamy residue on top and an almost clear fluid between. The essential finding in obstruction is food residue which may consist only of the rice and raisins of the night before or in addition food remnants of the day or even of three or four days before. Microscopically there may be mucus pus and particles of swallowed food. Starch is recognized by staining blue with iodine. Meat fibers look like small brown plaques with striations. Fat appears as globules which stain red with Sudan III. In the presence of blood staining by the Papanicolaou method may disclose cancer cells. When cancer is suspected a specimen obtained by abrading the gastric mucosa by means of a tube or bulb covered with gauze or a special abrasive brush will at times lead to the discovery of an early cancer. Late ulcerating cancer will usually shed typical cells which will be obtained with ordinary aspiration.

*Fractional gastric analysis* is done after the overnight residue has been removed. I make sure that the stomach is empty by having the patient swallow a few ounces of water several times and withdrawing it until the return is clear. The patient is then given 300 cc of water as a motor meal and an intramuscular injection of 0.33 mg of histamine acid phosphate as a secretory stimulant. After this at fifteen minute intervals 5 to 8 cc of content are withdrawn through the Rehfuess tube and placed in a row of bottles. Each specimen is titrated for free and total acidity, tested for occult blood and observed for bile color and visible blood. After one and one half to two hours the tube is removed. Formerly we inflated the stomach with air at the end of the examination while inspecting, palpating and percussing it to determine the size and height of its greater curvature. Our charts have room for recording all these findings as well as the ease of passing the tube and the condition of the patient during the test. Such a method of study gives valuable information about the curve of acidity, gastric motility and the nature of admixtures as follows:

1. The *type of acid curve* (see Fig. 34) (a) the *normal curve* showing an orderly rise and fall of acidity, (b) the *absence of any free hydrochloric acid* called *achylia* or *achlorhydria* and (c) the *reflex curve of continued hypersecretion* which does not descend during the second hour. Diminished or absent acidity may be found in gastritis and malignancy. The reflex curve is found in severe local irritation as in ulcer or some reflex cause of irritation as in appendicitis or pelvic disease. Normal or low normal curves may occur in gastric ulcers contrasted with the high reflex curves in duodenal ulcer. Mixed with blood they may also be found in gastric cancer.

2. The *motility or emptying time* of the stomach is tested by determining the amount of water given at the beginning of the procedure. At

rhage or operation diseases of the throat esophagus lungs or mediastinum especially aortic aneurysm and severe upper spinal curvature or arthritis

**THE GASTROPHOTOR** This tiny camera obscurely inserted into the stomach under fluoroscopic control and taking multiple films by direct intragastric illumination looked promising for a time. It has been generally abandoned by those who were most enthusiastic about it at first.

**THE STRING TEST** First described by Einhorn the test consists in having the patient swallow a yard long silk string or tape keeping it in one place overnight and studying it upon its removal in the morning. Stains indicating blood and bile are supposed to show the location of bleeding points. It was shown to be subject to so many errors that this method of examination has also been abandoned.

**Diet Study** A careful history of dietary habits is essential and should include the relation of food in general to symptoms whether causing, aggravating or relieving them the relation of specific foods to definite symptoms and the effect of changes in diet. At times a careful record by the patient of all details in regard to food intake and its relation to symptoms may be of great value. Experimental diets such as allergy diets may give valuable information.

**X ray Diagnosis** The x ray examination is the most important part of the examination on the stomach although its findings must nearly always be confirmed by a careful history and by fractional gastric analysis. Any discrepancy calls for repeated study.

**Fluoroscopy** is of definite though limited value. It is done first with the patient standing. Before giving the barium mixture the lungs heart and great vessels diaphragm and bony structure should be observed and films taken if any trouble is suspected. In the abdomen the arrangement of air and gas can be studied aided by plain scout films. The barium should be used first to study the esophagus as previously described. In the stomach as the opaque meal enters it the amount and consistency of gastric residue can be observed. The barium will not mix with thick mucus but will trickle through or infiltrate more liquid contents. The character of the rugae can be studied. Retention in an ulcer crater on the posterior wall can often be made out. Observation of defects or protrusions is aided by moving the patient about so that different aspects of the stomach can be seen. Peristalsis abnormal spasms and narrowings stiffening of the stomach wall and external pressure can be evaluated. At the pylorus and cap spasms obstruction and defects may be discernible. Prolapse of gastric mucosa into the cap may be observed. Diverticula may show up clearly. Mucosal irritability from allergy to a constituent of the barium meal is often striking. Examination in prone and supine positions may clarify findings seen in the standing patient and elevation of the foot of the table may demonstrate a hiatus hernia.

second hour may occur earlier as a result of nervous retching or of retrostalsis from a lesion elsewhere perhaps impinging on the gastrointestinal tract. Blood may be present in the first specimens as a result of trauma and will occur throughout in cases of gastric ulcer or neoplasm. The absence of blood in any specimen is evidence against an ulcerating lesion. Blood not present at first but appearing with regurgitation of bile from the duodenum is seen in ulcerating duodenal lesions and rarely in bleeding from the biliary tract. The finding of cancer cells has been mentioned. It is therefore readily seen that gastric analysis should be done in every case of suspected upper gastrointestinal disease.

*Tubeless gastric analysis* as a term is a misnomer. It refers to the use of ingested ion exchange resins to determine whether hydrochloric acid is being secreted in the stomach. In the presence of free hydrochloric acid the attached ion commonly quinine, methylene blue or azure A is displaced by the hydrogen ion of the acid and is excreted in the urine. The finding of quinine or the aniline dye in the urine indicates that acid is being secreted. This test has been used in screening out achlorhydric persons in mass surveys so that they can be further studied for pernicious anemia or cancer. However, as cancer can occur in the presence of free hydrochloric acid, it is not reliable as a cancer detection method. It can never replace adequate fractional gastric analysis which gives so much more valuable information than merely the status of the secretion.

*Uropepsin excretion* in the urine, considered an index of peptic activity and particularly of acid secretion in the stomach, has shown such wide variations even in normal subjects that its determination cannot be considered of sufficient value to justify its inclusion among standard tests.

*Instrumental Examinations* These are not of as much value as in the study of the esophagus. The mere passage of a stomach tube may, even counter deliver as a result of narrowing or spasm at the cardia. The distance to which it passes before striking the greater curvature gives an idea of the size and position of the stomach. At times the tube can be felt to pass rapidly into the duodenum, confirmed by immediate aspiration of bile indicating gastric hypermotility.

**GASTROSCOPY** Gastroscopy easily performed and with fair safety by an expert gives information about the appearance of the mucosa of ulcers or other lesions within view of the end of the instrument. However, the regions in which x-ray diagnosis may be confusing, the extreme cardiac and pyloric ends of the stomach and parts of the lesser curvature are frequently also outside the visual field of the gastroscope. Lesions below the surface of the mucosa cannot be seen. The newer operating gastroscopes are useful at times in obtaining small biopsy specimens from which a diagnosis is possible. Photographs made through the gastroscope are of limited value. *Contraindications* to gastroscopy include general debility or marked dyspnea from any cause, recent gastric hemor-

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*Radiography* may be done with spot films when fluoroscopy shows interesting lesions but it is better to take complete films after placing the patient in the proper position by using fluoroscopy as a guide. A number of films in various positions supine oblique lateral and prone enhance the value of the study. In this way not only are greater areas of the stomach brought into view but also its relations to the spine and other organs can be established. Rugal patterns defects protrusions foreign bodies tumors and the presence of hourglass or pyloric stenosis may be clearly demonstrated. After operations the patency of an anastomosis can be determined and defects protrusions fistulas and obstructions at or near the stomach can be diagnosed. With the use of extreme care by an expert practically all gastric lesions should be definitely demonstrable. Careless or inept technique will not only result in poor x-ray diagnosis but also may result in a patient's prolonged disability or even death.

It is important to realize that in any case of gastrointestinal symptoms even though they are thought to be due primarily to some gastric disturbance nothing short of a complete gastrointestinal x-ray study is fair to the patient or satisfactory to the clinician whose responsibility it is to take care of the patient. A routine series should consist at least of films taken two six twenty four and forty eight hours after the first gastric films. In some cases more frequent films may be indicated as for instance the hourly films taken for a study of the small intestine. No examination is complete without a barium enema study and cholecystography. Too many patients after incomplete x-ray films have been taken are told *nothing is the matter with them* and later are shown to have had a definite lesion that should have been demonstrated at the time of the first examination. Others are subjected to unjustifiable operations upon the stomach or other organs when an inoperable incurable lesion elsewhere had been overlooked through carelessness. I firmly believe that no roentgenologist should do only partial studies such as an immediate and six hour study a barium enema study or a cholecystography but should insist upon a complete series for the benefit of his own reputation and what is more important for the benefit of his patient.

*Summary* The diagnosis of gastric disease as has been brought out in this discussion must be made from a careful history not only of immediate symptoms but also of all antecedent conditions and operations any of which might have a bearing on the present situation. Accurate reports of previous operations reports and actual films of previous x-ray or laboratory studies reports from clinicians or specialists in various fields who have seen the patient must all be correlated. The information obtained must be used as a guide to the studies which must be made to round out a complete diagnosis. Only when a diagnosis has been

definitely established can the patient's physician expect to carry out a rational treatment and give a reasonably accurate prognosis

### TREATMENT

The treatment of diseases of the stomach must of course depend upon the nature of the disease. The general principles of *prophylaxis* in regard to hygiene and sanitation and the cure of general diseases removal of focal infections and *attention to diet* laid down in the first chapter must be borne in mind

#### Medicinal Treatment

Medicinal treatment also fully discussed in the first chapter should be confined to emergencies and to conditions in which specific therapy of some kind is indicated such as specific infections and infestations and cardiovascular renal pulmonary or pelvic diseases. Acids antacids enzymes sedatives hypnotics analgesics cholinergics and the many other forms of medication recommended for the stomach all aiming to correct functional disorders are best avoided until a definite diagnosis has been made. Many diseased conditions are amenable to relief or cure by physiologic or mechanical treatment alone. At times hormones may be indicated such as thyroid pituitary or estrogenic hormones or corticosteroids. In some diseases immunologic therapy may be of value. In the presence of a marked anemia hematinics or even transfusions may be necessary. The latter should however be avoided as much as possible because of the danger of unexplained reactions or the transmission of hepatitis. In incurable disease such as cancer any medication promising relief of symptoms should be used.

#### Physiologic Therapy

Physiologic therapy is most rational and also most successful. When a disease interferes with normal physiologic processes and results in symptoms treatment should be directed to the correction of abnormal and the re-establishment of normal gastric physiology. This is most frequently accomplished by simple dietary measures. It must be a rule that all diets no matter what their other requirements may be must consist of a normal balance of essential ingredients. Adequate protein carbohydrate and fat with sufficient vitamin and mineral content added if necessary must be provided. In planning diets it is of course essential to avoid any foods to which the patient is allergic. The temperature of the food is not important except that too hot foods irritate the stomach. The proportion of indigestible residue must be regulated according to the patency of the pylorus. The quantity and frequency of feedings must be regulated according to need.

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should get a feeding of fully 1 pint of food such as milk egg bread and cereal which will usually stay down. If not the process can be repeated. If vomiting follows again it is a good indication of actual obstruction lower down. Lavage is not advisable after operation on the stomach.

A sringe or mere aspiration through a stomach tube is of value in stenosis. At times it may be useful in gastritis if the stomach is filled with thick mucus. Its continued use is not justifiable.

*Electrotherapy* Electrotherapy in the form of galvanic or faradic current to stimulate peristalsis is no longer used.

*Radiotherapy* Radiotherapy by means of x-ray radium or radioactive elements such as cobalt 60 is of some use in the palliative treatment of gastric cancer. The best effect although not usually curative is produced in some forms of sarcoma.

### Psychotherapy

Psychotherapy has been used extensively especially in peptic ulcer. The claims made for psychoanalysis and prolonged discussions between psychiatrist and patient have been much publicized but the results of such special treatments are usually disappointing. In a sense psychotherapy is an essential part of all medical treatment. It is really the art of medicine. It takes into consideration the patient's stresses and strains and suggests measures for their elimination or alleviation. The most important factor in helping the patient psychologically is to make him confident that everything has been done to determine exactly what is the nature of his illness to describe it in detail and to explain the reason for each step in the treatment. This encourages the patient to follow instructions and to get the benefit of the treatment prescribed. In a case of cancer I do not inform the patient of it but usually speak of ulcer and its treatment. I believe that only in exceptional cases of incurable cancer should the patient be told.

### Surgical Therapy

Surgery has become increasingly safe with the really remarkable improvement made in techniques. Unfortunately this fact has been all too frequently made an excuse for unnecessary or ill advised operations. The ethical experienced surgeon does not rush into operations without careful evaluation of the indications for operation. Essentially operations on the stomach must be confined to (1) surgical emergencies such as injuries perforations ("the acute abdomen") or uncontrollable hemorrhages (2) complications such as obstruction walled-off perforation symptom producing adhesions to neighboring organs or congenital anomalies (3) attempted removal of neoplasms benign or malignant (4) postoperative complications such as obstructions hemorrhages perforations or fistulas.

Exploratory operations are in general a sign of failure in diagnosis.

ulceration a soothing more or less liquid diet is indicated. When disease has resulted in secretory disturbances such as reduction or cessation of acid secretion the diet should be bland and should contain no elements such as meat requiring the action of the acid for digestion. In such a case the administration of dilute hydrochloric acid is physiological in that it constitutes a stimulus to intestinal digestion. When a disease such as ulcer increases the secretion of acid foods which readily combine with acids are indicated and should be given at frequent intervals. If motor disturbances are produced such as the exaggerated hunger contractions associated with ulcer frequent feedings of adequate quantities of soothing foods will relieve or prevent pain. When the stomach is atonic because of nutritional or other constitutional disturbances a balanced stimulating diet with added vitamins and also with frequent feedings is indicated. When there is hemorrhage from the mucosal surface or a bleeding vessel the use of a food mixture containing the coagulant gelatin given in sufficient quantity and with sufficient frequency to keep the stomach at rest is most beneficial. In stenosis frequent feedings by mouth or tube of concentrated high caloric liquid foods with aspiration once or twice in twenty four hours of any residue remaining in the stomach improves the tone of the gastric muscles while also improving the nutrition of the patient and stimulating intestinal function. Parenteral feeding may be required when a patient is unconscious or mentally unbalanced or refuses to take food by mouth. Adequate balance of ingredients being impossible such feedings must of course be used only temporarily. Instead of attempting to prescribe special mixtures for parenteral use readily available solutions made by drug houses containing protein amino acids sugars vitamins and minerals can safely be used. Fat emulsions are also available.

### Physical Therapy

Physical therapy is not of much value in stomach disorders. External application of cold or heat may have some value as an analgesic but is mainly a psychological treatment. The same is true of massage. Heliotherapy has a good general tonic effect. Hydrotherapy externally even at spas is also mainly psychological in its effect. Ingested water in adequate amounts is a necessary part of any diet but can be overdone.

**Lavage** Lavage although at times of great value is not used as much as formerly when it was overdone. Autolavage is of use in persistent vomiting of reflex origin especially after operations. It is accomplished by having the patient drink rapidly at one time a full pint of warm water or hypertonic saline solution (1 or 2 heaping teaspoonfuls of salt to a pint of water). This is usually vomited immediately or within fifteen minutes. If it is vomited the same solution should be repeated at once. Fifteen minutes later whether vomiting has occurred or not the patient

starved dehydrated anemic patient is a decidedly poor operative risk. Two to three weeks of a nutritious well balanced diet with between meal feedings will improve the patient's general condition and gastric tone. I like to use a diet such as my ulcer diet (p. 267). If a stenosis is present aspirations, lavages and frequent liquid feedings by mouth or parenterally will be a great help. The other treatments mentioned in the paragraph on emergency operations should also be carried out. Immediately before operation some sedation is desirable although a patient who has been properly indoctrinated with a feeling of confidence and anticipation of worthwhile results is easy to handle.

**The Operation.** At operation when the conditions found are demonstrable a surgeon often finds it of value to consult with the clinician as to the most desirable operation to be performed. The question whether palliation or attempted cure with increased risk should be chosen must be decided upon. Though speed of operation is desirable too much haste may damage the tissues. Careful deliberate operations if they take a little longer are today safely performed and produce best results.

**Postoperative Care.** This starts as soon as the operation is completed. In some clinics a transfusion or parenteral glucose and saline solution is routinely given to prevent or control shock. The danger of upsetting the fluid and electrolyte balance must be borne in mind. One or at most 2 liters of parenteral fluid in the first twenty-four hours is usually sufficient and 5 per cent dextrose or levulose in normal saline solution is generally safe. Indicated cardiac stimulants, insulin or other medication may be given parenterally at first. Proper pulmonary ventilation should be encouraged at first by inhalation of carbon dioxide and oxygen. Later by the use of the blow bottle and deep breathing exercises. Pulmonary congestion is thus avoided and venous thrombosis prevented. The bladder should be carefully watched for urinary retention. The stomach should be immediately encouraged to function. Continuous suction with an indwelling tube introduced through the nose while helpful to prevent overdistention of the stomach is too often overdone and carried out for too long a time. It increases the danger of upsetting electrolyte and fluid balance, it has a bad psychological effect and it causes irritation of the nasal passages often with profuse discharge and the danger of respiratory infection. Many patients, especially those with some degree of nasal obstruction, prefer an orally introduced tube. Early feeding will usually obviate the use of suction altogether as can be shown if the tube is left in and occasional mild suction applied to check on the amount of fluid in the stomach. Presence of food in the stomach encourages peristalsis, improves gastric tone and stimulates intestinal function.

**PREVENTION OF POSTOPERATIVE GASTRIC DISTENTION OR ILEUS.** Feedings should be given preferably by mouth, otherwise through a tube.

## 232 Different Parts of the Gastrointestinal Tract

Of course all operations are in a way exploratory and a large incision is of help in such exploration. Except in emergency, however, no patient's abdomen should be opened until every diagnostic method has been used to determine every lesion in the abdomen. Rarely if ever should a surgeon be surprised at his findings during the operation.

**Preoperative Care** This is most important. Even in emergency operations certain preliminary procedures may be lifesaving. Careful general physical examination to evaluate the patient's general condition is essential. An x-ray scout film may give evidence of perforation or obstruction. Stool examinations will be a help. Examination of vomitus or aspirated gastric content may show blood or malignant tissue. A diabetic should be given insulin and glucose; a cardiac case may need a rapidly acting stimulant. Fluid and electrolyte balance must be attained by parenteral injections. Transfusion may be indicated; is in fact of value in any emergency operation and can be continued during the operation. Sedation must be cautiously administered. If possible the patient's drug and food idiosyncrasies should be learned. Prophylactic use of antibiotics or sulfa drugs is often of value. The anesthesiologist must choose his anesthetic with great care. The surgeon should not attempt too extensive an operation in emergency. It is safer to operate later if some corrective operation is indicated.

In *elective surgery* or in cases in which no immediate operation is indicated, the time spent in preoperative care is rewarding in reducing operative mortality, in permitting more complete operative procedures, in hastening convalescence and in preventing postoperative complications. Early or late operation should not be performed until a complete study has been made. When the abdomen is to be opened, it is imperative that all conditions which might influence the choice of operation or cause complications should be known to the surgeon. There should be a complete gastrointestinal study with gastric and stool analysis, x-ray studies consisting of a gastrointestinal series, barium enema and cholecystography, sigmoidoscopy, and when indicated gastroscopy. A careful check of cardiovascular, respiratory, urological and pelvic organs should be made. Adequate blood, urinary and other laboratory examinations should be carried out. Endocrine disturbances should be ruled out. Focal infections should be eradicated. Psychological factors should be checked. When abnormalities are found they should be eliminated or thoroughly treated. General hygienic measures should be instituted with proper rest and recreation, exercise, fresh air and sunshine. Care of the skin, especially on the abdomen, will help ensure good wound healing. Some surgeons like to give antibiotics before operation, but they may cause complications.

Adequate attention to *nutrition* is of the utmost importance. The

asionally allergy to certain foods may start after operation and cause diarrhea. This will require allergy studies (see p. 89).

5 Anemia as a result of loss of the gastric "intrinsic factor" is often troublesome and may require transfusions in an emergency. Otherwise the usual treatment for pernicious anemia is indicated. Vitamin B<sub>12</sub> either alone in large doses or combined with the expensive nucleoproteins or nucleopolysaccharides is the treatment used today. (See pernicious anemia in the section on Gastritis p. 231.)

6 Symptoms ascribed to reflux of bile and pancreatic juice into the stomach, the reflux of gastric contents into the proximal (afferent) loop of a gastroenterostomy, and combinations of these include retrostaltic symptoms even to the point of vomiting. They can be avoided by careful surgery and early and careful feedings such as have been described.

7 Symptoms such as those just mentioned together with a full feeling after eating attributable to the small size of the remaining gastric pouch are also prevented or treated in the manner described.

8 The "dumping syndrome" in which there occur episodes of nausea with or without vomiting, flushing, sweating, palpitation, abdominal distention, cramps, weakness and faintness has been much discussed and its occurrence has been attributed to various causes. It usually comes on soon after rapid eating or the swallowing of solid food and may be relieved by lying down. It has been suggested that hypoglycemia may be the cause of the symptoms. I have not seen any patient with these symptoms when the routine suggested above is followed. In my experience I have encountered it in cases in which feedings were not started early, in which too large feedings were started too late and in which the patient was permitted solid food, especially meat, too soon. When the syndrome occurs it is usually relieved by starting the diet as outlined immediately after operation and making additions as suggested.

9 Serious complications such as hemorrhage, acute cholecystitis or intestinal obstruction, partial or complete, may be relieved by measures suggested elsewhere for such conditions but frequently may require reoperation.

10 The late complications, marginal or jejunal ulcer, will be discussed under Ulcer, as will gastrojejunocolic fistula and jejuno-gastric intussusception.

### Anomalies of the Stomach

Congenital anomalies are exceedingly rare. Such conditions as atresia at the cardia or pylorus, absence of stomach, various degrees of nonrotation, inversion, transposition, megagastric, microgastric and double stomach have been described. Some are undoubtedly missed because infants are rarely examined by x-ray. As most of these anomalies tend to shorten life, few are found in adults.



every two or two and one half hours while the patient is awake. Normally they should consist of 4 to 6 ounces of milk, cream, glucose and gelatin mixture (see Stenosis Mixture p 276) or a solution of one of the powders prepared for tube feeding (e.g. Nutragest) fortified with vitamins and minerals. After the first thirty six to forty eight hours, cereal, soft eggs, cream, cheese, puddings, custards, Jello or ice cream should be added until in four or five days the patient is on an ulcer diet (see p 267). To this should be added gradually vegetables and fruits until a diet similar to the Gastritis Diet (p 252) is attained with no meat for six or eight weeks. Six feedings a day should be the rule. When much of the stomach has been removed and gastric digestion impaired, meat may not be well borne for months although the patient otherwise can be on a varied, well balanced diet.

*Postoperative Complications* Immediate or early complications include the following:

1 Vomiting best controlled by pushing feedings, repeating them if vomited. Autolavage or aspiration and lavage are indicated rarely, followed immediately by feedings. If prolonged vomiting occurs, parenteral feedings must be continued. Chlorpromazine should be used with caution because it may cause toxic hepatitis.

2 Acute dilatation of the stomach and duodenum and of the intestines generally was formerly an all too frequent complication following any type of major and even minor operation. Its exact cause was never fully understood. It was attributed to excessive fermentation, to a weakened gastrointestinal musculature and to neurologic imbalance. The patient with great abdominal distention, regurgitating gastric juice, often mixed with bile, becoming disoriented or delirious and with more or less febrile reaction was a fearsome spectacle. Gastric lavages, parenteral saline and glucose solution and efforts to induce expulsion of gas were of benefit. Autolavage, the enforced drinking of a pint of hypertonic saline solution (a heaping teaspoonful of sodium chloride in a pint of warm water) and its repetition if vomited, would in the absence of actual obstruction usually clean out the stomach sufficiently to permit of feedings. In severe cases surgical Pylritin or physostigmine intramuscularly combined with abdominal hot stupes and stimulating enemas was often successful. Today with the prophylactic treatment outlined above, postoperative ileus is rarely encountered and even milder distention is avoided. The subject is also discussed in the chapter on the Intestines (p 310).

3 Constipation usually due to insufficient food intake is generally prevented or relieved by the feedings and measures just mentioned for distention. Retention oil enemas of 4 to 6 ounces of warm vegetable or mineral oil will usually soften hard scybala or impactions.

4 Diarrhea may at times occur from excessive glucose in the feeding mixture and can be controlled by reducing or eliminating the sugar. Oc



Figure 32. Anomalies of stomach. *a* Band obstructing prepyloric region (1) fundus (2) corpus (3) antrum (4) narrowed pyloric region (a) cap. *b* Mucosal prolapse (1) antrum (2) duodenal cap (3) mucosa protruding through pylorus causing defect.

scarred obstruction from a healed ulcer or a beginning cancer. Figure 35 shows a partial obstruction by a band which produced symptoms when the patient had a cecal carcinoma. When symptoms of obstruction are present, operation is indicated.

#### HYPERRUGOSITY OF GASTRIC MUCOSA

Hyperrrugosity of the gastric mucosa, in which the rugae are much larger and coarser than normal, has been described as a congenital anomaly, although usually it has been considered due to chronic gastritis of the hypertrophic variety. It has been observed by gastroscopy and x-ray in persons who have had no gastrointestinal symptoms and no evidences of any other gastrointestinal disturbance. The mucosa, while coarse, showed no evidences of inflammation or other disease. This condition will be discussed under the subject of Chronic Gastritis (p. 250).

#### PROLAPSED GASTRIC MUCOSA

Prolapsed gastric mucosa through the pylorus is fairly common. It may occur spontaneously under normal conditions. It may be the result of loose redundant folds of gastric mucosa being washed or pushed through the pylorus into the duodenum. The loose attachment of the mucosa may be a congenital defect or may occur as a result of chronic irritation or inflammation. It has been shown to occur frequently in cases of hypertrophy of the pyloric muscle. It may result from traction on the mucosa when a tumor attached to the mucosa, most frequently a polyp, is pushed through the pylorus. As a rule, the mucosa moves in and out of the duo-

## HYPERTROPHIC PYLORIC STENOSIS

The *congenital form* occurs in less than 0.5 per cent of all births mostly in firstborn male children and especially in twins. It is supposed to be a familial anomaly and has been attributed to an allergy. It is usually not recognized until the persistent forceful vomiting unlike the usual initial regurgitation is recognized by the mother or friends as being abnormal. Usually from two to four weeks or longer have elapsed the infant has lost weight and the stools and urine have become scanty. Examination shows a distended stomach with visible peristalsis followed by vomiting of food with no bile admixture. The hypertrophied pylorus may be felt as a small spool like tumor especially when the stomach is empty. X-ray examination may show a stomach alternately atonic and hyperactive with evidence of obstruction at the pylorus the degree of which will determine the amount of emptying. The stomach may show retention for twenty-four hours or more but some barium and air will go through the narrowed pylorus. The condition may be confused with pylorospasm which is normally observed in older children and duodenal atresia in which no barium or air is seen beyond the stomach. A burn from ingestion of corrosive chemicals or a scirrhus pyloritis may cause confusion but are much more rare. Treatment consists essentially in attempting small frequent feedings, parenteral feedings and daily lavage and antispasmodics especially atropine methylnitrate (a few drops of Metopine Elixir before feedings). Many cases will respond to this type of therapy. In persistent cases surgery is uniformly successful and the mortality rate low. The Rammstedt operation or one of its modifications consisting in longitudinal section of the thickened muscle without injury to the mucosa is the most successful procedure.

Hypertrophic pyloric stenosis has been described in the *adult*. It is usually attributed to disease such as ulcer, gastritis or reflex pylorospasm. However there have been reported typical cases resembling those found in infancy in which the patients usually men have given a history of gastric complaints since childhood accompanied as a rule by attacks of vomiting and pain. In such cases the x-ray findings at the pylorus have often been considered due to malignancy until resected specimens have shown the characteristic hypertrophy. The treatment would be the same as that for pyloric stenosis due to ulcer, cancer or other cause.

## STENOSIS DUE TO CONGENITAL BANDS OR ADHESIONS

Stenosis due to congenital bands or adhesions is a rare anomaly. It will cause symptoms similar to those present in other forms of stenosis. Partial obstruction may continue into adult life when some condition causing irritation or spasm of the pylorus may produce symptoms even complete obstruction. The diagnosis by x-ray may be confused with

Protrusion of prolapsed mucosa upward into the esophagus has been observed but is an extremely rare condition

#### TREATMENT

If no symptoms are present usually no treatment is required. If other lesions are found these should be treated medically or surgically in the usual way. If symptoms are apparently being caused by the prolapse and more or less irritation and thickening of the mucosa is present an ulcer diet will usually be of great benefit. As a rule surgery is not of benefit unless complications such as hemorrhage or obstruction require it. Pyloroplasty would be the operation of choice. Subtotal gastrectomy has been done by some surgeons but seems to be too radical a procedure.

#### CASCADE STOMACH

This term is applied to a variation in form and shape of the stomach in which the fundus lying posteriorly becomes a pouch because the corpus and antrum lie farther forward and on a higher plane than normal. The contents of the fundus are pushed over the hump and cascade into the corpus like a waterfall. As a congenital anomaly it occurs usually in persons of the *hypersthenic* habitus the wide broad type in whom the stomach naturally lies almost in a transverse plane. As an acquired deformity it may be caused by external pressure upward against the greater curvature by a distended colon by an enlarged pancreas by perigastric adhesions or retroperitoneal tumors. Eventration of the left dome of the diaphragm may produce a similar effect by pulling the fundus upward and backward. Gastric lesions such as ulcer carcinoma or cicatrices may also produce a similar appearance.

#### SYMPTOMS

There are few symptoms directly attributable to the cascade stomach. In the congenital type the stomach has usually so adapted itself to the condition that only under stress will symptoms occur. Aerophagia with its feeling of epigastric or lower sternal fullness or pressure when the stomach becomes distended with the swallowed air is a common accompaniment. The distress relieved by belching recurs frequently with repeated air swallowing. With the belching there may be some regurgitation or actual vomiting of gastric content. In the organic type the symptoms of the causative factor will usually overshadow any symptoms caused by the cascade.

#### DIAGNOSIS

There are no characteristic symptoms and the physical findings are variable. The x ray therefore remains the only method of diagnosis. At times fluoroscopy or films of the abdomen without use of the opaque meal

denum fairly freely without being damaged but at times it may become incarcerated and be difficult to reduce even at operation. The protruding mucosa may be edematous or thickened causing more or less obstruction or may become irritated and eroded producing hemorrhage. If a polyp is present this may ulcerate and bleed or may undergo malignant degeneration.

### SYMPTOMS

Most cases are free of symptoms and are discovered in the course of a gastrointestinal study which discloses some other definite lesions accounting for symptoms. Studies of large groups of patients in whom no other lesion was found have not revealed any characteristic manifestations; the patients usually complaining of retrostaltic or reflex symptoms including fullness, heartburn, air swallowing, belching, nausea, regurgitation and vomiting with resultant loss of weight and strength. There may be epigastric pains immediately after eating not relieved by food or alkalies and coming on suddenly in attacks which may last only a short time. In the intervals between attacks there may be entire freedom from pain and even from the reflex symptoms. Many such cases are mistaken for ulcer, gallbladder disease or any other gastrointestinal disease and may in fact accompany these conditions. When medical or surgical care has resulted in demonstrable cure of these concurrent lesions and symptoms persist mucosal prolapse must be considered one of the possible causes. When the pylorus becomes obstructed the characteristic symptoms of pyloric stenosis will occur.

### DIAGNOSIS

The prolapse can be recognized only by x-ray examination and of course only at the time it is actually present. The prolapse may at times be induced by pressure over the stomach or by having the patient bend or otherwise exercise when the stomach is full of the opaque mixture. Multiple films in different positions and with varying degrees of gastric filling may be necessary to establish the diagnosis. The characteristic umbrella or mushroom like defect produced by the protrusion of the gastric mucosa into the base of the duodenal cap when once recognized by the examiner will rarely elude his watchful eyes in subsequent examinations (Fig. 35 b). The differentiation from a neoplasm either a polyp or cancer is usually fairly easy especially when the prolapse is reduced and the defect it causes disappears. A careful mucosal study will usually disclose a polyp if present. If the prolapsed mucosa becomes wedged into the pylorus causing obstruction the usual findings of pyloric stenosis will be demonstrable. In such a case a review of former films made of this patient's stomach may disclose a prolapse which had been overlooked and will be a help in deciding the cause of the obstruction.

Protrusion of prolapsed mucosa upward into the esophagus has been observed but is in extremely rare condition

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ligaments and perigastric adhesions may be contributing factors. Colonic diverticulitis and pancreatic malignancy have been blamed. It may follow persistent vomiting, marked retrostibis or acute gastric dilatation. In marked rotation over 180 degrees the onset is usually acute, the symptoms suggesting an acute abdominal calamity. Vascular occlusion may occur with resultant shock and necrosis. An x-ray scout film may be of help in ruling out perforation or intestinal obstruction. Immediate operation is indicated. Reduction of the volvulus may be attempted at once or reserved for a second operation after a preliminary enterostomy. The mortality rate is high.

In less acute cases in which there may be no symptoms, volvulus must be differentiated from a cascade stomach. After a barium mixture the volvulus will show two fluid waves, the cascade one. In volvulus the rotated greater curvature forms a convex curve continuous with and simulating an enlarged sweep of the duodenum. In cascade stomach the greater curvature is lowermost, as in the normal stomach, and the esophagus enters the fundus normally, whereas in volvulus it is low, owing to rotation. Operation is indicated for volvulus. In partial volvulus occurring in the distal portion of the stomach and usually due to tumors or ulcers with hourglass or pyloric stenosis, the volvulus may not be recognized as a part of the abnormal findings, but as surgical intervention is indicated, it will be discovered at operation.

#### DIVERTICULOSIS

*True or congenital diverticula* not uncommon in other parts of the alimentary canal, are rarely encountered in the *stomach*. When present they are usually on the posterior wall near the lesser curvature, just below the entrance of the esophagus where the musculature is weakest, permitting the protrusion. They are usually single, rarely have two or more, been found. They are more frequent in females and are usually not discovered until middle life. Association with esophageal, duodenal and colonic diverticula is not uncommon. True diverticula are small pouches, usually round, and varying in size from 0.5 to 4 cm. in diameter (Fig. 37 a). They should show no signs of intrinsic or extrinsic disease to account for their origin and should contain all coats of the stomach wall, although the muscular coat may be very thin. Gastric diverticula rarely become inflamed or ulcerated and even more rarely may be the site of malignant changes. Being usually small and retaining little gastric content, they seldom cause any symptoms. A larger one may cause slight dysphagia, vomiting or epigastric pain after eating. With ulceration, hemorrhage may occur, with inflammation there is pain, fever and tenderness.

*Duodenal diverticula* are much more common and may be found in any portion of the duodenum and further down in the small intestine. They





*Figure 36 Cascade type of stomach. Postero anterior view illustrates the cardia of the stomach beneath the left diaphragm the body and pylorus being situated in the midline and to the right (Ickman Clinical Roentgenology of the Digestive Tract 3rd ed.)*

will show the large air bubble at the fundus with some air in the corpus (Fig 36). The barium meal will usually show the condition sometimes brought out better after only a little opaque mixture. A careful study with multiple films must be made and it is extremely important to rule out an organic cause before regarding the case as one of congenital cascade stomach. Differentiation from a volvulus is discussed in the next section.

#### TREATMENT

No specific treatment is indicated for the congenital type. Lying on the right side after a meal will facilitate emptying of the pouch. The diaphragm should be abolished as has been described elsewhere by explaining the mechanism to the patient and insisting that breathing through the open mouth when the full feeling comes on will prevent air swallowing. A normal diet with between meal and bedtime feedings avoidance of laxatives and proper hygienic care are indicated. In the organic cases the underlying cause must be treated.

#### VOLVULUS

Volvulus in which the whole or part of the stomach becomes rotated on its long axis is an extremely rare condition. It may occur as a result of lengthening of the stomach and relaxation of its supports. It has occurred as an accompaniment of diaphragmatic hernia eventration or paralysis. Tumors or cicatrices in the stomach tears of its supporting

a healed ulcer or by tumors. Their pouches may be larger than the true diverticula and may be irregular in shape and mucosal outline. The symptoms are those of the causative factor although a large retentive or inflamed diverticulum may aggravate these symptoms. X-ray diagnosis may be difficult because of the findings connected with the cause. Differentiation from the crater of an ulcer or ulcerating carcinoma depends upon the finding of the smooth pouch with its neck at the gastric mucosa contrasted with the crater of the ulcer which may be smooth or irregular but which is surrounded by an area of defect due to induration or infiltration. A perforated walled-off ulcer will also show this induration and in addition is most frequently located on the lesser curvature far from the usual location of the diverticulum. Esophageal diverticula and hiatus hernia are easily differentiated. The treatment is that of the cause and is usually surgical especially if malignancy is suspected. In the rare complication of perforation of a diverticulum symptoms and treatment are the same as for perforation elsewhere.

#### HERNIA OF THE STOMACH

Hernia of the stomach through the hiatal orifice has been discussed under esophageal conditions as has the high position of the stomach in eventration of the diaphragm.

#### PANCREATIC ANOMALIES AFFECTING THE STOMACH

These include annular and aberrant pancreas. They are discussed in the chapter on the Pancreas (p. 588).

*Annular pancreas* in which the pancreas surrounds the duodenum is also a confusing anomaly. It may cause no symptoms unless it becomes swollen with disease when it may occlude the duodenal lumen. It is not easy to recognize by x-ray study.

Pancreatic rests or aberrant heterotopic collections of pancreatic tissue may rarely be found in the stomach or duodenum and may cause no symptoms. Occasionally they may be mistaken for ulcer or neoplasm. They may produce symptoms resembling those of ulcer or cancer, may cause severe bleeding and in x-ray films usually are mistaken for polyps or cancer. They are usually not accurately diagnosed except at operation or necropsy.

### Diseases of the Stomach

#### GASTRITIS AND DUODENITIS

The term "gastritis" is a misnomer and for a long time there was a good deal of controversy about the name. If gastritis means inflammation of the stomach and if inflammation always means infection the name would apply to but a small proportion of the so-called gastritides. A

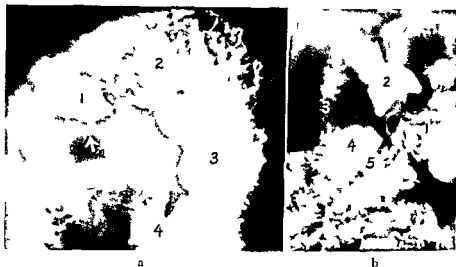


Figure 37 a Large diverticulum of stomach near cardiac orifice (1) (2) fundus (3) corpus (4) antrum b Diverticulum of duodenum third portion (1) antrum (2) cap (3) third portion of duodenum (4) diverticulum (5) orifice of diverticulum

may be single or multiple may vary in size from 0.5 to 10 cm in diameter. They usually fill and empty readily so that rarely are they the cause of symptoms. Pain and retrostaltic and obstructive symptoms have been attributed to them but seldom proved. Inflammation, ulceration, malignant changes, hemorrhages and even perforation of diverticula have been reported. They are usually found during the course of a routine gastrointestinal x-ray study and can be differentiated from walled off perforated ulcers by their smooth outline and fairly well defined orifice. Unless complications have developed no specific care is indicated.

*Diagnosis* is made by x-ray films taken in different positions and at different angles preferably standing when the pouch can be seen smoothly filled with barium (Fig 37 b) sometimes also with air producing a fluid level. Retention of barium in the pouch at six or even twenty-four hours or more may call attention to a diverticulum missed in the initial examination. They may be transient seen only on repeated x-ray studies. Differential diagnosis is discussed below. Unless complications are demonstrable or causing symptoms no treatment is required. If distress is present postural drainage may relieve it.

*Acquired gastric diverticula* unlike true diverticula may be caused by sudden increased intragastric pressure the so called pulsion type or more frequently by extragastric conditions the traction diverticula. The latter may be due to traction from adhesions to neighboring organs or lymph nodes usually is a result of inflammation in these organs or the stomach, the former from weakening of the stomach wall by the scar of

### Food Poisoning

Food poisoning in which usually not only the stomach but also the intestines are involved in an acute gastroenterocolitis may result from infected fermented or essentially poisonous food or from food to which the patient is allergic. The symptoms primarily vomiting as a rule with cramps diarrhea and prostration may start immediately after the offending food has been ingested or may not appear until hours or days later. A careful history of food ingested and of possibly infected food handlers is important. Cultures and chemical study of vomitus feces and any remnants of suspected foods is important in making the diagnosis. Eosinophilia may be found in allergic gastritis.

The *treatment* consisting essentially in eliminating the offending food by induced vomiting lavage and catharsis preferably by castor oil as well as medication and antibiotics is described in the chapter on Food Poisoning in general (p. 71). In most cities such cases must be reported to the health department for epidemiological study.

### Corrosive Gastritis

In this disease the mucosa and occasionally also deeper layers of the gastric wall become necrotic as a result of the ingestion of corrosive substances. The esophagus is first affected to a variable degree followed by corrosion of the stomach. Some corrosives will stop in the stomach others notably bichloride of mercury will pass through the pylorus causing corrosion in the small and even the large intestines. The corrosives which may have been taken accidentally or with suicidal intent include such substances as strong acids alkalis corrosive sublimate (bichloride of mercury) phenol iodine or lysol. All will produce burns which may go on to necrosis sloughing and ulceration and at times may cause hemorrhages or even perforation. Healing may be followed by more or less deforming scars or strictures. Other less severe corrosives with less effect during their rapid progress through the esophagus include potassium chlorate silver nitrate oxalic acid phosphorus arsenic and other insecticides. "Black leaf 40" 40 per cent nicotine sulfate will cause such rapid death from absorption that its corrosive effects are of minor importance.

*Complications* in other organs may be serious. Excretion of poisons through the kidneys may cause serious damage the patient dying in uremia as in bichloride poisoning.

The *symptoms* consist in extreme pain caused by the corrosive action vomiting of the corrosive which will increase the effect on the esophagus and mouth bleeding and later diarrhea often bloody followed by marked prostration or collapse.

*Examination* will usually show evidence of corrosive action on the lips and in the mouth and throat. The abdomen is tender and, if perforation

broad interpretation of the meaning of the term includes under it not only inflammation but also irritation of the gastric mucosa and its sequelae. Included under the term are irritation from physical agents such as heat and trauma chemical and food poisoning changes due to hormonal and endocrine disorders and mucosal reactions due to allergy and to malignant disease. It must be borne in mind that rarely is the stomach alone affected by many of these etiologic factors but that more or less disturbance occurs not only in the duodenum but also along the whole length of the alimentary canal. With this understanding we can go on and discuss the various types of gastritis and duodenitis without confusion. As in any other diseases it is important to determine the cause of the condition and the treatment must consist primarily in the removal or treatment of the cause. Prophylaxis consists in avoiding the various etiologic factors mentioned above. In the following discussions of gastritis it must be understood that in most cases the duodenum is also affected but the term *gastroduodenitis* is not being used because it is too cumbersome.

### ACUTE GASTRITIS

Any of the factors just mentioned may be the cause of acute gastritis. The type and severity of the acute symptoms the methods of diagnosis and the treatment vary with the cause. Following is a simple classification of the different types.

#### Simple Acute Catarrhal Gastritis

Catarrhal gastritis is better called acute endogastritis since usually only the mucosa is involved in the catarrhal inflammation. As the cause is usually a swallowed irritant the esophagus and usually the duodenal cap will show the same effects of irritation. The irritation may come from swallowing excessively hot liquids or large amounts of irritating liquids such as coffee tea and spices or condiments or alcohol or the mere overeating of any food especially poorly masticated coarse foods. Similar effects may result from drugs such as iodides bromides salicylates quinine creosote chloride and many others even including sodium bicarbonate to excess. Such a comparatively mild gastritis may cause only mild symptoms of epigastric distress fullness heartburn nausea regurgitation or less frequently vomiting and diarrhea. These symptoms are usually of short duration and may disappear within a few hours or a day or two after the cause has been eliminated. A careful detailed history of all food ingested will usually determine the cause of the disturbance.

*Treatment* should consist in eliminating the cause and taking a bland smooth diet (see Ulcer Diet p. 267) for a few days and instructing the patient to avoid future attacks.

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Complications in other organs may be serious. Excretion of poisons through the kidneys may cause serious damage, the patient dying in uremia as in bichloride poisoning.

The symptoms consist in extreme pain caused by the corrosive action, vomiting of the corrosive which will increase the effect on the esophagus and mouth, bleeding, and later diarrhea, often bloody, followed by marked prostration or collapse.

Examination will usually show evidence of corrosive action on the lips and in the mouth and throat. The abdomen is tender and if perforation

is impending symptoms of an "acute abdomen" will be found. Instrumental and x-ray examinations are usually contraindicated but examination of vomitus and stools will show bloody mucus and shreds of mucosa. Specimens of each together with glasses or containers from which the victim has drunk should be sent at once to a toxicological laboratory.

*Treatment* consists in giving antidotes for the corrosive poison before inducing vomiting. Extreme care in passing a stomach tube to avoid perforation. The ingestion or instillation of emollients such as oils or gelatin solutions and early feeding. The diet should at first be liquid as in esophageal stricture. Later smooth bland foods are gradually added as the lesions heal. The esophageal lesions must be cared for (p. 198) often by dilatation as described in the chapter on the Esophagus. Renal complications require suitable treatment. Gastric deformities or pyloric stenosis resulting from cicatricial contraction may require operation.

### Acute Infectious Gastritis

This disease occurring in the course of acute generalized infectious diseases such as pneumonia, virus infection, typhoid and others which may also cause a general gastrointestinal disturbance is considered to be due to actual secondary infection of the stomach wall or to allergy followed by infection. The *symptoms* may be mild and hardly noticeable or severe with epigastric distress, vomiting and diarrhea as the worst features. They may be sufficiently severe to indicate need for a gastric analysis which may show hypochlorhydria as a result of fever, sometimes with occult blood. X-ray films usually show nothing abnormal except some coarsening of gastric rugae and perhaps pylorospasm and intestinal hypermotility. The *treatment* should be for the original infection plus a bland or ulcer diet during the course of the disease.

### Acute Phlegmonous Gastritis

This disease also called suppurative or purulent gastritis is usually due to hematogenous infection in septicemia, endocarditis or infectious disease. The infection may come from some distant suppurative focus such as osteomyelitis, cystitis or erysipelas or may be due to secondary infection in cancer or ulcer or following trauma to the stomach. It is a rare condition associated with gangrene of the stomach wall with severe *symptoms* resembling those of an acute abdominal calamity. The diagnosis is therefore rarely made until the condition causing the symptoms is discovered at operation. The outcome is almost invariably fatal.

### CHRONIC GASTRITIS

Chronic gastritis has been a controversial diagnosis over the years. Before the advent of modern diagnostic methods it was a scapegoat

diagnosis applied to any gastric disturbance not clearly understood. With the beginning of a new diagnosis of modern gastrointestinal surgery and the finding of ulcer, cancer and other lesions, gastritis was completely overlooked; its presence in fact was denied and the diagnosis ridiculed. Then the use of the gastroscope, permitting direct visualization of the mucosa, resulted in large numbers of enthusiastic observers rushing into print with elaborate classifications of the various types of gastritis which they had seen. Literally dozens of varieties were described and pictured. The enthusiasm became somewhat dampened when Wolf and Wolff and other careful observers, studying the gastric mucosa of patients with a large gastric fistula, described the mucosal changes occurring during emotion and stress, changes in temperatures and upon ingestion of various foods and chemicals. The different pictures of the normal mucosa taken under these various conditions resembled almost all the findings which had been described as due to different varieties of gastritis. It is therefore important today not to place too much emphasis upon the gastroscopic diagnosis of gastritis. The old simple classification of chronic gastritis as of two varieties, the hypertrophic and atrophic, with a "mixed" type consisting of some features of each, is gradually being favored by authorities, but even this classification is under criticism.

The "*mixed type*" is a simple endogastritis with little or no involvement of submucosa as a rule, but perhaps with areas of atrophy of the mucosa and other areas of hyperplasia or hypertrophy.

In the *atrophic type* the mucosa and often the deep layers have become thin and atrophic with more or less destruction of secreting cells. It is in this type that there is supposed to be a reduction or entire disappearance of acid secretion and even of enzymes. Many such stomachs, however, show normal acid curves on gastric analysis.

In the *hypertrophic type* there is proliferation of the mucosa, increase in size of the lymph follicles and more or less fibrosis of the deeper layers. The rugal folds usually become thickened and coarsened. Acid secretion may or may not be impaired. In some cases the mucosa becomes so coarsened and the rugae so large and deep that they are described as "giant rugae." These are usually confused with the hyperrugosity which is supposed to be a congenital anomaly. With the latter, however, there are no evidences of inflammatory or fibrotic changes. It must again be emphasized that the changes observed in the stomach are usually also present in the duodenal cap so that the term *gastroduodenitis* might be preferable. When the term *gastritis* is used we must understand that *duodenitis* is included.

The various other classifications of gastritis have been the result of (1) observing the appearance of the mucosa and describing erosive, ulcerative and granulomatous gastritis; (2) considering the location of the mucosal changes such as antral, fundic or pyloric gastritis; a pan-



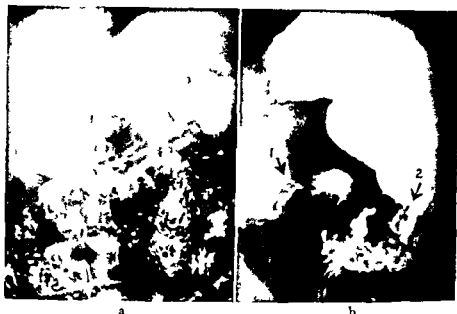


Figure 38 *a* Hyperplasticity of gastric mucosa. Note small lumen and irregular but smooth mucosal pattern. *b* Antral gastritis due to duodenal ulcer showing (1) duodenal ulcer (2) coarsened rugae gastric antrum

**Stool Examinations** Stool examinations are of some value. After a diet containing meat the finding of many undigested meat fibers suggests an acidity, although of course it is also due to deficiency of pancreatic secretion which must be ruled out. Finding occult blood after a meat free diet for four days is of only questionable value since the blood may come from anywhere in the alimentary canal even as a result of trauma to the gums from a toothpick. Examination for ova and parasites may disclose information of value.

**Blood studies** are necessary. As a rule in gastritis there is present more or less secondary hypochromic anemia. The relation of pernicious anemia is well known. Eosinophilia may be found in an allergic gastritis.

### Diagnosis

The diagnosis of chronic gastritis should never be made until all other causes of symptoms especially cancer have been carefully ruled out. The history of prolonged abuses of diet and medication of general and gastrointestinal chronic diseases of hormonal and endocrine disturbances and of allergies elsewhere may lead to a suspicion of chronic gastritis. The findings on x-ray gastroscopic examination and fractional gastric analysis will usually give evidence of the existence of this condition.

### Treatment

*Prophylactic* treatment is most important. It should include (1) avoidance of all causative factors especially alcohol and foods or drugs to which the patient is allergic; (2) attention to normal dietary habits; (3) proper care of gastrointestinal function in the course of acute or chronic diseases and after operations as discussed below.

*Indications for Treatment* Treatment is indicated in the following cases:

1. When gastritis is *secondary* to an *intrinsic gastric or duodenal lesion* such as ulcer, cancer or vascular disease, these require appropriate care which will also benefit the gastritis.

2. When due to constitutional disease, vascular disease or parasites, attention to these conditions is essential.

3. Avoidance of all known gastric irritants such as alcohol, coffee, tea, drugs, tobacco and agents to which the patient is known to be allergic is important.

4. When excessive amounts of *mucus* cause discomfort, nausea or vomiting, lavage with normal saline solution or sodium bicarbonate once or twice a day may be used as a temporary expedient, but as a rule is unnecessary. When marked stasis occurs in patients with stenosis, aspiration or lavage may be indicated as described under treatment for ulcer or neoplasms.

5. If *hemorrhages* occur, the treatment should be that recommended for patients with hemorrhages from ulcer, the diagnosis being postponed for a week or two until hemorrhage has stopped.

6. In the *absence of acid secretion* (achylia), dilute hydrochloric acid, formerly much recommended, is now rarely used, even when so-called gastrogenous diarrhea is present. Avoidance of meat and uncooked starch in the diet, because the protein envelopes of meat fibers and starch granules require hydrochloric acid for their digestion, will usually control the diarrhea. If diarrhea persists, however, in spite of the physiologic treatment recommended below, dilute hydrochloric acid in doses of 30 to 60 minims (2 to 4 cc.) will at times stop the diarrhea. To prevent dental erosion, the acid should be dissolved in one half glass of sweetened water taken through a tube, one half hour after meals, and should be followed by a mouth rinsing. One of the preparations which release acid in the stomach, such as Normacid, is equally effective and nonerosive.

7. *Anemia* calls for treatment. The microcytic hypochromic (*secondary*) anemia requires only iron therapy by mouth. Macrocytic hyperchromic (*pernicious*) anemia, due to the loss of the intrinsic factor, is today treated by vitamin B<sub>12</sub>. The dosage of B<sub>12</sub> needs to be stabilized by frequent blood and bone marrow studies, even as the dose of insulin is governed by blood and urine studies. If given alone by mouth, a large initial dose of 5 mg. followed by 1 mg. (1000 micrograms) or less per

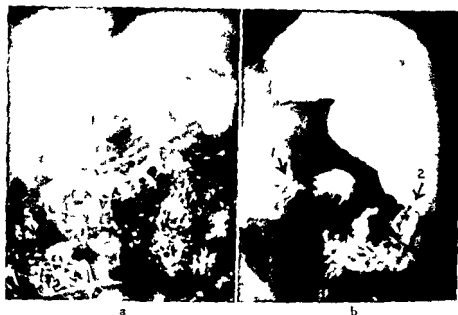


Figure 38 *a* Hypergastrinemia of gastric mucosa. Note small lumen and irregular but smooth mucosal pattern. *b* Antral gastritis due to duodenal ulcer showing (1) duodenal ulcer (2) coarsened rugae gastric antrum

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patient is shown to be allergic principally by causing diarrhea (*b*) the occurrence of diarrhea independent of allergies by reducing fats and oils fruits vegetables or excessive sugar as required (*c*) the presence of constipation by addition of larger amounts of the foods just mentioned (*d*) meats added cautiously after six to eight weeks watching for signs of irritation in the stomach and bowel

### Prognosis

The average case of gastroduodenitis treated as outlined will show much clinical improvement within a short time. Subsequent studies of gastric secretion however will rarely show return of hydrochloric acid even though the patient is doing well and eating nearly everything. With the knowledge that restrictions on meat are particularly desirable the patients are usually happy with the results.

### GASTRIC AND DUODENAL ULCER

The term "peptic ulcer" for either gastric or duodenal ulcer is commonly used and is a convenient general term especially when the exact location of the ulcer is not known. It is open to the objection however that it implies the ulcer has been caused by the action of pepsin, an old theory that has been definitely disproved. It has been held that ulcers have been getting more common but this contention is probably not true the apparent increase being due to improvements in diagnosis. Fifty years ago the diagnosis of ulcer was made principally from the symptom of hemorrhage and was considered a disease of young girls. With improvements in diagnostic methods especially the x-ray ulcers have been more and more frequently recognized but even today many cases are missed because most ulcers heal rapidly and spontaneously leaving in many instances almost unnoticeable scars. Patients often recover before a diagnosis can be made.

### PATHOLOGY

Ulcers while usually single may be multiple. Two or three generally in different stages of healing may at times be seen in the stomach and cap or in both at the same time. At autopsy many scars may be encountered at times recognized only by microscopic examination of serial sections. Ulcers usually occur along the lesser curvature the pylorus and on the anterior and posterior walls of the cap. They may be large or small but large ones up to 2, 3 or 4 cm in diameter are most frequently found along the lesser curvature smaller ones down to the size of small caplets are in the pyloric region and cap. Though a few benign ulcers of the greater curvature have been observed it is safer to consider all ulcers as malignant. Ulcers of the second portion of the duodenum are rare. Jejunal or marginal ulcers occur after anastomotic

week is the average dose. Intramuscularly 20 to 30 micrograms per week and later perhaps per month will be sufficient. Tablets of vitamin B<sub>12</sub> in smaller doses but combined with the intrinsic factor nucleoprotein or nucleopolysaccharide are orally effective but expensive over long term use. The unfortunate indiscriminate use of small doses of vitamin B<sub>12</sub> and folic acid and liver extract for all anemias serves to disguise true cases of pernicious anemia without controlling the serious neurological involvements and is to be condemned.

**Physiologic Treatment** This consists in soothing the irritated gastric mucosa, attention to gastrointestinal motility and adequate nutrition. For these purposes medication is usually of no particular value. Demulcents such as oils or gums taken alone may cause nausea and provoke diarrhea. Gelatin is of most value in this respect and is not only soothing but also is a protein food which promotes coagulation of blood. It should be continued in a soothing, well balanced nutritious diet. As mentioned above meat and uncooked starches are avoided when hydrochloric acid is absent but may be tried cautiously when symptoms have subsided usually after six to eight weeks. Such a patient should be admonished *never to take meat to excess thereafter and preferably to avoid it entirely* two to three days a week. In my experience the best results are obtained from a diet such as the gastritis diet. This diet not only is of value in the treatment of patients with known gastritis from any cause but also should be used for the prevention of postoperative gastritis. It should be started a few days after the liquid diet which is preferably used immediately after operation on the stomach and upper gastrointestinal tract and is also of value in cases of operation outside the gastrointestinal tract.

### Gastritis Diet

<i>Breakfast</i>	Milk 1 glass
	Cereal 4 to 8 ounces with milk and sugar
	Eggs 1 or 2 boiled or poached
	Bread and butter (or toast) 1 slice
	Fruit cooked, canned, strained or juice
<i>Luncheon</i>	Milk 1 glass
	Egg (1) or cheese (mild) 1 ounce
	Vegetables all kinds—thoroughly cooked, strained at first
	Lettuce—corn or peanut oil with lemon juice
	Bread and butter 1 slice
<i>Supper</i>	Pudding, Jello, ice cream, fruit (cooked or strained)
	Same as luncheon
<i>Between meals and at bedtime</i>	Gelatin powdered unflavored 1 heaping teaspoonful mixed in water or milk
	Milk 1 glass
Vitamins and minerals in the form of standard capsules should supplement this diet	

It must be understood that this diet will require modification in respect to (a) the patient's allergies by avoiding any foods to which the

slough cut or may be eroded by the gastric juice when it digests out the necrotic area resulting in *hemorrhage*. The extent of the *infiltration* occurring about an ulcer and the degree of accompanying peritoneal reaction are dependent upon the size and particularly upon the depth of the ulcer. *Stenosis* resulting from cicatricial contractions after healing occurs mostly at the narrow pylorus. It is much less common than stenosis resulting from perigastric and periduodenal adhesions caused by *perforations*. These include so-called slow perforations into neighboring organs principally the head of the pancreas or the liver perforations covered by omentum and perforations into the gallbladder or transverse colon with fistula formation.

The reason for the occurrence of the necrotic areas constitutes the most important causative factor in the production of ulcer. This fact has gradually come to be recognized, and many explanations have been advanced. The fact that ulcers occur along the lesser curvature and the duodenum can be explained by the fact that there is an end artery circulation in this region. That some ulcers occur as a result of obliteration of these end arteries due to arteriosclerosis, obliterating endarteritis, polyarteritis nodosa, embolism or trauma is undoubtedly true but these are rare causes as is syphilis. That vasospasm resulting from psychic causes could proceed to actual death of tissue seems highly improbable although the occurrence of ulcer following psychic trauma has been attributed to this cause. That body habitus should play a part is incomprehensible and is belied by the fact that ulcer is seen proportionately as often in patients of asthenic as of sthenic habitus. Some endocrine disturbances associated with vascular abnormalities could undoubtedly play a part in causing arteriolar spasm if not obliteration. *Rauwolfia* preparations used in the treatment of hypertension as well as other drugs lowering blood pressure have been shown not only to cause exacerbations in patients with a history of previous ulcers but also to cause ulcers in patients without such a history. Ulcers have also been attributed to Butazolidin and to corticosteroids. Allergy which is known to cause necrotic areas and ulceration in other parts of the body such as the skin, conjunctiva and the buccal and colonic mucosa must be considered an important etiologic factor although peptic ulcer due to food or drug allergy is rare. The fact that ulcers start as necrosis in the submucosal areolar tissue also suggests their inclusion among so-called collagen diseases.

In 1912 and 1913 two research workers made contributions of great importance to the etiology of ulcer. Rosenow demonstrated the connection between focal infection and ulcer previously suggested by Frank Billings. Turk produced typical gastric ulcers in dogs by intravenous injections of simple extracts of homologous tissue attributing the resulting necrotic areas in the stomach and elsewhere to absorption of a

operations on the stomach. Anatomically true ulcers must be distinguished from the erosions usually multiple which occur with gastritis. Surrounding an ulcer there is generally a ring of induration apparently formed to protect the ulcer from trauma and to restrict its motion. More or less mucosal reaction (gastritis) may be present not only around the ulcer but also throughout the stomach and cap. Ulcers are frequently associated with other diseases of the gastrointestinal tract and elsewhere notably with liver and gallbladder disease, appendicitis, colitis and coronary artery disease.

## ETIOLOGY

### Pathogenesis

The old persistent idea that peptic ulcers are caused by erosion of the mucosa by acid gastric juice is gradually but surely being abandoned as careful study and research reveal its fallacy. This theory was based on the fact that excess secretion of hydrochloric acid has usually been found in the stomachs of patients with ulcer ignoring the fact that some ulcers especially those in the stomach are found in the presence of an achlorhydria. The fact has been ignored that ulcers heal spontaneously or even when dilute hydrochloric acid has been given owing to a mistake in diagnosis. In general the increased acid secretion can be explained on the basis of irritation of the gastric mucosa caused by the presence of ulcer. The spontaneous healing of the ulcer in the presence of the increased acidity could be attributed to the cleaning out of the base of the ulcer by the acid promoting the healing process. That the increased acid secretion is a protective reaction to the presence of the ulcer is a much more rational explanation than that it is a destructive agent.

Normal gastric mucosa is protected from digestion by gastric juice so that it is necessary to presuppose damage or actual death of a localized area of mucosa before the juice could digest it out and produce an ulcer. Lewis Gregory Cole in his carefully checked pathologic researches demonstrated conclusively that ulcers do not originate in the mucosa but like boils and carbuncles originate as necrotic areas in the deeper layers of the stomach wall. Later breaking through the mucosa the size of the resulting ulcers depending upon the depth and extent of the necrotic area. Large necrotic areas such as those occurring along the lesser curvature of the stomach with its loose submucosal tissue will produce the large ulcers seen there where small ulcers are found in the prepyloric and duodenal regions rarely beyond the duodenal cap. When necrotic areas are deep and occur in regions where the wall is thin and the lumen small particularly in the duodenum the breaking through may occur in both directions through mucosa and serosa at the same time producing acute perforation with peritonitis or chronic perforation with adhesions. When a vessel happens to lie in the sloughed area it may

taneous healing. During the intervals between typical attacks of demonstrated ulcers x ray examinations and even operations have disclosed either that no ulcer was demonstrable or that at times only scars of healed ulcers were present. In different attacks recurrent ulcers may be found at various points in the stomach and duodenum. Thirty years ago Lewis Gregory Cole demonstrated the rapid and complete healing of uncomplicated gastric ulcer at first by frequent roentgen studies and later by detailed pathologic research. Later studies have shown conclusively that uncomplicated duodenal ulcers will also heal completely although at times leaving deformities from scarring because of the smaller lumen of the duodenum.

One fact in the ulcer problem that stands out prominently is that no matter what type of treatment has been used, the reports show that ulcers have healed as demonstrated by x ray and clinical findings before and after treatment. Whether the patient has been treated by antacid, hormonal or endocrine therapy, whether he has been fed through the mouth or nose into the stomach or duodenum or even through the veins or rectum, whether the foods have consisted mainly of carbohydrates, fats, proteins or amino acids, whether psychotherapy, physiotherapy or radiotherapy have been applied, and whether any one of the various operations ranging from simple gastroenterostomy, pylorotomy or gastrectomy to the "psychosomatic operations" of vagotomy or prefrontal lobotomy have been performed, the reported results are approximately the same. The old familiar statistic is inaccurate when applied to clinical reports in that it does not show the value of each of these methods of treatment. A more complete picture may be obtained by a rational, unbiased perspective. The conclusion that uncomplicated ulcers must heal is true, but the variation in healing time by different treatments is due to the fact that some treatments interfere with others.

### SYMPTOMS

The most important point in the history of the "periodicity" of symptoms. A diagnosis of ulcer is made from the history of attacks of indigestion, pain, or vomiting alternating with periods of entire well-being, regardless of diet or activity. This sequence of attacks and subsequent healing of ulcers is characteristic of no ulcer is present. This history will be found in the history. With marked deformity from scarring, the symptoms persist in the interim period. With a large ulcer, adhesions, there is usually little if any



theoretic substance from dead tissue which he called "cytost." Later he called attention to the fact that "cytost" was undoubtedly present in small quantities in the necrotic tissue in so called foci of infection and that its absorption could duplicate his findings after the injection of "cytost" in dogs. This was further emphasized when it was realized that Rosenow in his animal experiments injected mixtures of the infective material from foci of infection and not pure cultures of the bacteria into his animals' veins so that cytost was included. The absorption of cytost would also explain the Curling ulcer following burns and the ulcers occurring after bad fractures, other trauma or operations. Whether cytost is the histamine like substance, perhaps serotonin, which various workers have been trying to implicate as the cause of ulcer, or whether the reaction in the mucosa is a so called auto allergic phenomenon due to sensitivity to bacteria or the products of bacterial action at areas of focal infection remains to be demonstrated. Forty years of experience in the eradication of all foci of infection in my patients with ulcer has constantly emphasized for me the importance of focal infections in the etiology of ulcer. I have never seen an ulcer occur in any case in which such foci could not be demonstrated as a cause. Recurrences which might be postponed for an indefinite period by thorough eradication of foci have practically always been found to be due to recurrence of old focal infections or the development of new ones. That some patients tend to have ulcers rather than other conditions known to be caused by focal infections would point to a constitutional tendency to the development of ulcer which seems to be hereditary.

That not all patients with evident focal infections have ulcer is no more an argument against focal infection as a cause of ulcer than the fact that not all persons inhaling dusts or pollens have asthma or hay fever.

### Psychic Influences

The idea that ulcers may be psychosomatic in origin is not borne out by my experience. Although severe psychic trauma may act as a trigger to initiate an attack of ulcer, I have found that invariably after a thorough eradication of all etiological factors mentioned above the same or more severe psychic trauma will not alone cause another ulcer.

### NATURAL COURSE

All uncomplicated peptic ulcers heal spontaneously and rapidly as has been amply demonstrated. One proof of this can be found in the typical history of ulcer, the so called chronicity and periodicity. The typical recurrence over a long period of years of ulcer symptoms (hunger pain relieved by food) with often long intervals of entire freedom from symptoms whether the patient has been treated or not indicates spon-

tinuous healing. During the intervals between typical attacks of demonstrated ulcers x-ray examinations and even operations have disclosed either that no ulcer was demonstrable or that at times only scars of healed ulcers were present. In different attacks recurrent ulcers may be found at various points in the stomach and duodenum. Thirty years ago Lewis Gregory Cole demonstrated the rapid and complete healing of uncomplicated gastric ulcer at first by frequent roentgen studies and later by detailed pathologic research. Later studies have shown conclusively that uncomplicated duodenal ulcers will also heal completely although at times leaving deformities from scarring, because of the smaller lumen of the duodenum.

One fact in the ulcer problem that stands out prominently is that no matter what type of treatment has been used the reports show that ulcers have healed as demonstrated by x-ray and clinical findings before and after treatment. Whether the patient has been treated by antacid, hormonal or endocrine therapy, whether he has been fed through the mouth or nose into the stomach or duodenum or even through the veins or rectum, whether the foods have consisted mainly of carbohydrates, fats, proteins or amino acids, whether psychotherapy, physiotherapy or radiotherapy have been applied, and whether any one of the various operations ranging from simple gastroenterostomy, pylorotomy or gastrectomy to the "psychosomatic operations" of vagotomy or prefrontal lobotomy have been performed, the reported results have all been approximately the same. The old familiar statistical studies so notoriously inaccurate when applied to clinical reports in general have been used to show the value of each of these methods of treatment. Looking at the whole picture any rational unbiased person could come to only one conclusion: that uncomplicated ulcers must heal spontaneously and that the variation in healing time by different methods of treatment *must* be due to the fact that some treatments interfere with healing more than others.

#### SYMPTOMS

The most important point in the history of ulcer is the "chronicity and periodicity" of symptoms. A diagnosis of ulcer can almost be made alone from the history of attacks of "indigestion" lasting from a week to a few months alternating with periods of entire absence of symptoms regardless of diet or activity. This sequence of events corresponds to the formation and subsequent healing of ulcers followed by periods during which no ulcer is present. This history will be modified if complications occur. With marked deformity from scarring or adhesions some symptoms may persist in the interim period. With a perforation walled off and causing adhesions there is usually little if any relief of symptoms.

## Pain

The pains in ulcer patients may be of all three types discussed in the general section on gastric physiology. The *hunger pain* due to exaggerated hunger contraction occurs when the stomach is empty or nearly empty. This pain usually in the epigastrium may be reflected upward downward to either side or to the back. At times especially when the ulcer is complicated by disease in other parts of the gastrointestinal tract the pain may be located in the gallbladder region the back (with *pancreatic irritation*) the appendix region or over the sigmoid. In any case it will occur late after eating (one to three or more hours) and will be relieved by eating. The time of occurrence of the hunger pain is independent of the location of the ulcer but is often directly due to the quantity of food ingested occurring earlier after small feedings later after large meals as would be expected. Hunger pains may be completely masked as a result of frequent feedings.

The second type of pain *due to pylorospasm* in the case of an ulcer in close proximity to the pylorus may occur immediately after or even during eating and is usually accompanied by retrostaltic symptoms such as nausea belching (aerophagia) sour eructations regurgitation or even vomiting. These retrostaltic symptoms may occur with any ulcer regardless of location.

A third type of pain *due to peritoneal irritation* occasionally occurs in large lesser curvature ulcers when the crater becomes stretched with gastric contents that is immediately after filling the stomach and will last until intragastric tension is relieved usually one half to one hour being succeeded later by hunger pain. Peritoneal pain also occurs in deep duodenal ulcers which are impinging against the peritoneum and is of course severe if the peritoneal coat is perforated.

*Persistent pain* little or not at all relieved by food usually indicates a complication such as a walled off perforation and adhesions to neighboring organs or actual disease in these organs (pancreas gallbladder liver omentum).

## Bleeding

Bleeding from the surface of an ulcer a slight oozing from the eroded mucosa occurs in all ulcers usually only in the early stages. Bleeding is not noticed by the patient unless it is of considerable degree when hematemesis and melena may occur. This will be discussed under Complications.

## Vomiting

Vomiting has already been mentioned as a retrostaltic symptom when it occurs immediately after ingestion of food or drink. In such a case the vomitus consists of the recently ingested material mixed with more or

less gastric juice. In the night or after any long period of fasting the vomitus may be sour or bitter when consisting of gastric juice and usually bile or may be tasteless "waterbrish" when consisting only of swallowed saliva. *Salivation* is a rare symptom occurring at the same time as hunger pains or only at night. *Delayed vomiting* occurring many hours after eating and consisting of food taken a long time before is indicative of stenosis and will be discussed under that complication.

### Secondary Symptoms

*Appetite* varies. There may be a voracious appetite or a more or less complete anorexia. Some patients who have not discovered relief by food may be afraid to eat and hence may *lose weight*. Others in efforts to relieve pain may eat large quantities of food and *gain weight*. With insufficient feedings consisting of soft food *constipation* may be annoying and patients may list this as a chief complaint. *Diarrhea* may result from excessive alkali ingestion or from allergy to some food in the diet, often milk. The severe diarrhea of jejunoileic fistula will be discussed later. *Weakness* and *pallor* may be due to an insufficient and unbalanced diet or to recent or recurrent hemorrhage. *Alkalosis* is not as frequently observed now as formerly when excessive doses of soluble alkalis were taken. *Nervous symptoms* are usually present as would be expected in a patient with the symptoms of ulcer occurring at intervals producing feelings of apprehension and insecurity. These symptoms will usually disappear with the healing of an ulcer and may recur with subsequent ulcers.

### PHYSICAL EXAMINATION

Only rarely are there any findings of importance on abdominal examination. There may or may not be *tenderness* in the midepigastrium. Tenderness may be elicited only at the location of a complication such as gallbladder disease, appendicitis or sigmoid diverticulitis or even outside the gastrointestinal tract.

*Muscle guarding* or rigidity is usually due to complications such as perforation or near perforation. Rarely in a thin patient an indurated ulcer may be palpable. A stomach distended with food and showing peristalsis will be found with pyloric stenosis. Definite pallor suggests bleeding.

A *general physical examination* is essential. A thorough search for focal infections in the mouth, nose, throat, sinuses, rectum, pelvis and prostate is most important. Careful chest, cardiovascular, renal, pelvic and neurological studies should be conducted. Rectal examination should not be neglected. Search for evidences of allergic, endocrine or hormonal conditions should be thorough.

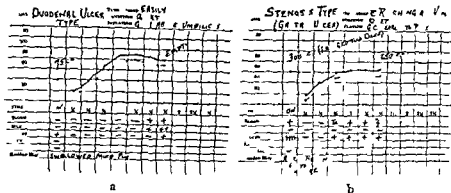


Figure 39 Fractional gastric analysis *a* Duodenal ulcer type Secretion reflex type of curve motility rapid emptying (usually) admixtures blood in regurgitated bile *b* Stenosis type Secretion high in residue rapid rise motility overnight retention large residue at end admixtures blood in specimens—gastric ulcer no blood—duodenal ulcer In fasting contents food remnants yeast and sarcinae

### LABORATORY EXAMINATION

Gastric analysis is of considerable value in many cases. Fasting contents removed the morning after the usual rice and raisin duration meal taken at bedtime if they contain visible rice and raisins indicate a stenosis either in midstomach (hourglass stomach) at the pylorus or in the duodenum. The quantity of overnight residue is somewhat an index to the degree of obstruction a large quantity with much liquid indicating a small lumen. The acidity may vary being usually lower in gastric than in duodenal ulcer. The microscopic findings include sprouting yeasts or sarcinae in the presence of free hydrochloric acid usually found in ulcer as contrasted with lactic acid bacilli and free lactic acid when no free acid is present as is often the case in carcinoma causing obstruction. Cytological study of fasting contents or of material obtained by means of one of the abrasive balloon or brush type of instruments may be of value in the diagnosis of cancer. In high grade obstruction the overnight residue will often contain remnants of food eaten days before.

Fractional gastric analysis will produce a curve of acidity which is not always characteristic. In gastric ulcer the acidity may be high approaching a free hydrochloric acid of 60 or 80 units but it may also be low or even absent in which case cancer must be suspected. In duodenal ulcer the curve of free acidity usually rises rapidly to high figures even to 90 or 100 or more and tends to remain high indicating hypersecretion usually associated with this condition (Fig. 39 *a*). Absence of blood in the gastric contents is contrary to the presence of an active ulcer but its presence may be due to trauma in passing the tube. If no blood is seen in the early removals but begins to be noticed toward the end of the examination after one or one and one half hours and coincident with regurgitation of bile into the stomach it is indicative of a source

of bleeding in the duodenum and will confirm the presence of an active duodenal ulcer which may have been only suspected by x ray examination. The persistent finding of blood may also be of help in determining the presence of a peptic ulcer marginal or jejunal ulcer. Delayed emptying combined with a large overnight residue indicates stenosis pyloric or duodenal (Fig. 39 B).

Stool examinations are of little value in ulcer. Occult blood found in many stools even on a strict meat free diet may be due to slight trauma or bleeding anywhere from mouth to anus or in any of the tracts opening into the gastrointestinal tract. Absence of blood may be an indication that no active ulceration is present and is of value in determining cessation of bleeding after massive hemorrhage. Immediately after such hemorrhage the stools are tarry.

Other laboratory examinations such as blood cell counts, serologic tests and the various tests for possible specific diseases causing ulcer will be of obvious advantage.

#### INSTRUMENTAL EXAMINATIONS

*Gastroscopy* in expert hands will show an ulcer if it comes within the limited area of the stomach which can be viewed by this method but errors in diagnosis are frequent. The new operating gastroscope designed by Benedict permits of biopsy so that differentiation from carcinoma is possible in cases in which the ulcer can be reached.

*Proctoscopy* should not be neglected. Aside from its value in ruling out malignant disease or a specific colitis the finding of fissures, fistulas or cryptitis is of importance from the standpoint of focal infection.

X ray examination is valuable if performed by an expert. To make a diagnosis of ulcer it is essential to identify a definite niche or crater filled with barium mixture surrounded by a defect due to inflammatory changes, edema or induration. This is only rarely possible by fluoroscopy alone, even large lesser curvature ulcers being easily overlooked. Serial roentgenograms are absolutely necessary and must be taken in different positions with and without compression in order to bring out small ulcers (Fig. 40). Too often a diagnosis of ulcer is made only from the presence of spasm or from a deformity in the stomach or duodenum. At times a crater not seen in the early films may be well visualized at one or two hours after the barium meal. It is important to recognize the difference between the smooth crater of an uncomplicated ulcer and the deeper, often spreading crater or extravasation produced by a walled off perforated ulcer usually in the duodenum which will often retain barium for six or more hours (Fig. 41). Films taken at two and six hours will show whether an obstruction is present. For x ray findings of complications see the appropriate heading below. In any case a complete x ray study with films at twenty four and forty eight hours

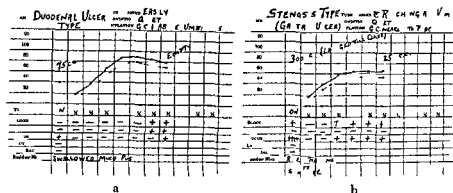


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Figure 41 Duodenal ulcer showing in *a* (1) crater of ulcer with cap compressed (2) pylorus (3) antrum in *b* (1) extra gasation due to walled-off perforation, (2) narrowed duodenal cap (3) antrum

a barium enema and a cholecystography are advisable to identify complications which might have a bearing on successful treatment. The section on X-ray Diagnosis shows details.

### DIAGNOSIS

The three essential diagnostic factors in ulcer described above are the following:

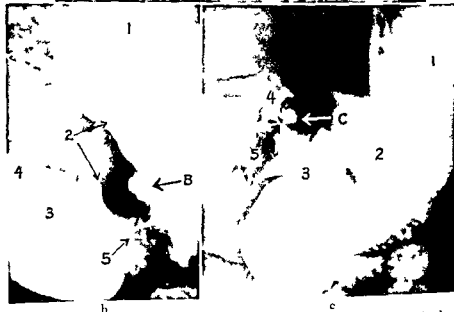
- 1 The *history* of typical *chronicity* and *periodicity* of symptoms which in most cases consists of the symptom complex of hunger pains relieved by any kind of food or drink and by alkalis. Retrostaltic symptoms while usually present do not differentiate ulcer from other diseases affecting the gastrointestinal tract. The significance of other pains and symptoms has already been discussed.

- 2 The *x-ray finding* of an ulcer crater. Though secondary findings such as spasms, incisuras, irregularities and motility changes may be due to ulcer, they are not diagnostic. The x-ray findings in complications such as perforation and stenosis are discussed below.

- 3 *Fractional gastric analysis* as has been mentioned is of value in demonstrating the continued secretion mostly found in duodenal ulcer as well as the finding of blood only in regurgitated duodenal contents. It is also of value in demonstrating a bleeding lesion (ulcer or cancer) or in ruling out such bleeding. Its value in hemorrhage and stenosis will be discussed later.



a



b

c

Figure 40 Gastric ulcer *a* Ulcer near cardia (at arrow *A*) showing (1) fundus (2) pars media (3) antrum (4) duodenal cap (5) spasm due to ulcer *b* Lesser curvature ulcer (at arrow *B*) showing (1) fundus (2) pars media (3) antrum (4) duodenal cap (5) spasm due to ulcer *c* Prepyloric (channel) ulcer (at arrow *C*) showing (1) fundus (2) pars media (3) antrum (4) duodenal cap (5) spasm due to ulcer

ter of fact gastric ulcers heal so rapidly that there is not time for malignant change to occur except in the scars resulting from healing and this is unusual. The fact that simple ulcers heal rapidly is the basis for making a re-examination of all gastric ulcers within three or four weeks after the finding of the ulcer. At this time the ulcer crater may be distinctly smaller or entirely absent and its surrounding induration diminished or gone as contrasted with increasing and irregular infiltration in the case of malignancy and increase and often irregularity in the contours of the malignant ulcer crater. A few cases have been reported in which the carcinomatous crater was smaller because of proliferation of the tumor tissue within the crater but careful observation would show an irregularity of outline at this site and the increased surrounding infiltration. Cytologic study of gastric contents inspection or even biopsy of the ulcer through a gastroscope inspection and handling of it at operation and even removal of the ulcer and its microscopic study may often not result in a definite diagnosis. It is only by actually observing that an ulcer is healing or has healed as shown by repeated x-ray studies that a carcinoma can be definitely ruled out.

#### MEDICAL TREATMENT

##### Prophylactic Treatment

In the treatment of peptic ulcer prophylaxis is of paramount importance. I have seen patients who when they had ulcer recurrences were scolded because they had deviated from a prescribed diet had failed to take medication had overexercised or had continued to carry on nerve-racking business affairs. I have never seen any of these restrictions prevent the occurrence of ulcers. As I mentioned before ever since relation between focal infection and ulcer was first demonstrated in 1913 I have carried out systematic and thorough eradication of all infective foci in the body and my results have justified the really great effort involved in carrying out this program. I have never observed the development of subsequent ulcers except in cases in which some new infection had developed or an old one had recurred and subsequent care of these would then give excellent results. Clinicians opposing removal of foci are invariably those who tried it but did not carry out a comprehensive program and therefore got no results. My contention is that a clinician allowing obvious infections to remain in any patient who suffers from any chronic disease whether it has been shown to be one due to focal infection or not is failing to give the patient adequate care that is he is treating a disease and not the patient. Such neglect is getting to be too common. The physician taking care of an ulcer patient should consult the section on Focal Infections (p. 19) and carry out in detail its recommendations for extraction of all nonvital or pyorrheic teeth and retained root fragments removal of infected tonsils adequate treatment of ear

## DIFFERENTIAL DIAGNOSIS

When the three diagnostic factors mentioned above are present the diagnosis is established. Other diseases may at times mask ulcer symptoms however resulting in neglect of the three methods of diagnosis.

In *gallstones* pylorospasm and retrostiltic symptoms usually immediately after meals have been mistaken for ulcer. The frequent occurrence of both *gallstones* and *ulcer* with the *hunger pangs* often referred to the gallbladder region may cause confusion but complete study should obviate this.

*Pancreatic disease* with pangs in the epigastrium and back is often differentiable by careful diagnostic study and the incidental stool and blood findings. Pancreatic "rests" in the stomach or duodenum may closely simulate ulcer.

*Allergic manifestations* especially erosive gastritis or duodenitis occasionally caused by tobacco coffee or other foods may produce temporary intermittent symptoms resembling those of ulcer but can be differentiated by careful study. Real ulcers may also be caused by allergy.

*Hypoglycemia* produces at times severe hunger sensations and should be looked for in difficult cases especially if the patient is a diabetic taking insulin. I have seen ulcers develop after severe hypoglycemic reaction.

*Psychic disturbances* may be associated with ulcer symptoms. I have seen patients become psychotic after having been treated for ulcer and develop an obsession for frequent feedings to allay gnawing pains.

The differential diagnosis between ulcer and *cancer* is vital. Duodenal carcinoma is rare and is usually associated with biliary tract or pancreatic carcinoma. It is therefore rarely confused with ulcer. A gastric ulcer on the other hand must always be suspected of being a carcinoma with ulceration until proved benign especially if the patient has no history of previous ulcers. An ulcer on the greater curvature is practically always malignant. The percentage of occurrence of carcinoma as compared with ulcer along the lesser curvature or on the posterior wall near the lesser curvature is not of great help in differentiation. The fact that most carcinomas occur between the incisura and the pylorus does not mean that an ulcer seen elsewhere is not malignant. Less than 4 per cent of patients with typical benign ulcer will be found later with cancer and only a few have been found with cancer developing in the ulcer or scar. There is no reason why in a patient with an ulcer history a cancer cannot develop later. Usually when the cancer develops a careful history will disclose a change of symptoms greater persistence and no remissions. No patient should be treated for ulcer merely because previous x-ray films have shown one. A complete study is always indicated. The patient's safety demands a definite diagnosis. The old teaching that gastric ulcer may become malignant is based on erroneous diagnosis of cancers from the start having been mistaken for simple ulcers. As a mat

ter of fact gastric ulcers heal so rapidly that there is not time for malignant change to occur except in the scars resulting from healing and this is unusual. The fact that simple ulcers heal rapidly is the basis for making a re-examination of all gastric ulcers within three or four weeks after the finding of the ulcer. At this time the ulcer crater may be distinctly smaller or entirely absent and its surrounding induration diminished or gone as contrasted with increasing and irregular infiltration in the case of malignancy and increase and often irregularity in the contours of the malignant ulcer crater. A few cures have been reported in which the carcinomatous crater was smaller because of proliferation of the tumor tissue within the crater but careful observation would show an irregularity of outline at this site and the increased surrounding infiltration. Cytologic study of gastric contents inspection or even biopsy of the ulcer through a gastroscope inspection and handling of it at operation and even removal of the ulcer and its microscopic study may often not result in a definite diagnosis. It is only by actually observing that an ulcer is healing or has healed as shown by repeated x-ray studies that a carcinoma can be definitely ruled out.

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nose and throat and sinus infections eradication of infections in the female pelvis the genitourinary tract rectum lungs bones or skin The neglect of even one small root fragment may cause a recurrence of ulcer

The elimination of all known *allergens* from the diet and avoidance of tobacco especially if the patient is allergic to it are of course essential Avoidance of such gastric irritants as coffee tea or other caffeine-containing drinks alcohol meats of all kinds especially meat extractives spices herbs and fried foods is of temporary value in ulcer patients

### Treatment of Uncomplicated Ulcer

Ever since ulcer was first recognized as an entity various treatments have been described until now there is a confusing profusion of therapies including all kinds of medications physical x ray and psychiatric treatments and surgical procedures At first and even today much of the treatment has been purely empirical or symptomatic not taking into account the accumulated knowledge about the etiology pathogenesis and natural history of the disease Empirical treatment consisted in neutralization of the acid gastric juice by alkalies antacids mucin and various colloidal preparations or preventing or reducing its secretion by means of anastomoses resections nerve blocks or anticholinergic drugs Surgical attack even now does not invariably include resection of the ulcer Symptomatic treatment has included sedatives antispasmodics antiemetics and demulcents Lavages intragastric and intrajejunal feedings parenteral injections of various kinds endocrine and hormonal cabbage and other juices and even x ray treatments have been used New drugs and new methods of treatment are appearing with bewildering frequency and an antiulcer vitamin has been postulated Authorities have differed as to the need for bed rest hospitalization or ambulatory treatment There have been differences of opinion as to dietary indications among equally eminent clinicians as to whether high protein high carbohydrate or high fat diets are best whether foods should be predigested whether roughage should be restricted or avoided and whether it is best to make no recommendations as to diet except for frequency of feedings

The surprising fact about the hundreds of treatments recommended is that practically all of them have been effective Their efficacy has been demonstrated by x ray films taken before and after treatment showing disappearance of an ulcer crater The reason is plain when we realize that all uncomplicated ulcers will heal spontaneously and rapidly being not only uninfluenced by treatments but often actually interfered with by treatments Careful studies of each new drug or method of treatment have consistently shown that no treatment has a specific effect upon an ulcer Many drugs especially anticholinergics may relieve pain and

mask symptoms. Serious complications even perforation may thus be overlooked.

### Instructions to Patients

1 It is of great value psychologically and to ensure cooperation in the treatment to show the patient a picture of an ulcer to indicate on the picture the location of the ulcer and to emphasize that if uncomplicated it will heal. It should be demonstrated by comparison with the ulcer following a boil in the neck that in healing there may be a little scarring or a marked deformity which if located at a narrow point may cause obstruction with characteristic delayed vomiting. Bleeding can be easily explained by pointing out that a vessel may be injured during sloughing out of the ulcer "core." Perforation can also be explained. It is important to show how ulcers originate beneath the mucosa "like a boil" as a result of a reaction probably allergic caused by a distant infection rarely by food or other allergy. With a patient so indoctrinated it is easy to treat him.

2 *Diet* The patient must be instructed as to the purpose of the diet. When he knows that pain occurs when the stomach is empty, he realizes the importance of frequent feedings and that the duration of the relief and therefore the frequency of the feedings depend upon the size of each feeding. Comparing the ulcer with ulcer following a boil explains the need for smooth "salvelike" foods. Since this "salve" leaves the spot and obviously cannot be kept in place by a bandage, continuous effect can be achieved only by frequent applications in the form of feedings. The patient must also be informed of the need for a nutritious well balanced diet to promote healing and since the diet may be low in vitamins and minerals, the need for vitamin and mineral supplements. The purpose of keeping the diet simple to avoid possible allergens and irritating or stimulating constituents is also easily explainable. Now the patient is prepared to understand and take his diet. One I have used for many years with few modifications is as follows:

### Ulcer Diet

#### *Breakfast*

- Milk 1 glass
- Cereal large dish with sugar and milk or cream
- Egg 1 or 2 soft boiled or poached
- Bread or toast with butter 1 or 2 slices
- Juice of 1 orange at end of meal

#### *Midmorning*

- Powdered gelatin 1 1/2 teaspoonful stirred into 1 glass of water taken quickly and followed by 1 glass of milk

#### *Luncheon*

- Milk 1 glass
- Egg soft boiled or poached or custard
- Baked or mashed potato or plain spaghetti

Bread and butter 1 or 2 slices

Pudding custard gelatin ice cream or stewed fruit

*Midafternoon*

Same as midmorning

*Supper*

Same as breakfast or luncheon

*At bedtime* and at 2¼ hour intervals in night if awake

Feedings as between meals

Vitamins and minerals can be provided with one of the commercial preparations

Mineral oil 1½ ounce (no more) at bedtime may be required because of lack of residue in the diet but should not be used routinely

Vegetable oil 1½ ounce just before meals is helpful in patients with marked gastric irritability and hypermotility

This diet is to be followed for six weeks. Its caloric value can be modified according to necessity. It may also have to be modified in accordance with food allergy. In case of allergy to milk, gelatin and soy bean preparations may be used (see section on Allergy, p. 87).

No medication is required or desirable. In the absence of complications this diet almost invariably will relieve ulcer symptoms at once or within a few days. If this relief is not forthcoming, some complications must be suspected, either of the ulcer itself, such as perforation or stenosis, or complications elsewhere, as discussed under Differential Diagnosis.

During the six weeks of this diet the eradication of all focal infections should be carried out. With each attack on a focus an exacerbation of ulcer symptoms may occur, but is usually of short duration unless a new ulcer has developed. In either case, continuance of the diet is all that is required.

After six weeks, vegetables and fruits of gradually increasing bulk content are added to the meals. Usually in three months, if all foci have been cleared up, a general well-balanced diet should be prescribed, but the between-meal feedings should be continued indefinitely.

With this regimen carefully followed, I have known patients to keep free of any ulcer attacks over long periods, up to twenty-five and thirty years or more. Almost invariably, recurrences of ulcer are due to exacerbations of old focal infections, such as sinus infections, or the development of new ones, such as devitalization of a tooth.

### SURGICAL TREATMENT

Surgical treatment is never indicated in uncomplicated ulcer. There is no operation which, in itself, will prevent the occurrence of more ulcers. The so-called intractable ulcer is always a complicated ulcer.

When operation has been decided upon, *preparation for operation* and *postoperative care*, as described in the general section on the stomach (p. 232), should never be neglected. It is only with careful attention to all details that a good result can be assured. Poor results can usually be traced to their neglect.

## COMPLICATIONS OF ULCER

The complications of ulcer requiring special care include hemorrhage perforation and stenosis. Formerly "carcinomatous degeneration" was included among the complications but as pointed out before it is extremely rare errors in the past having been due to mistaking a carcinomatous ulcer for a simple gastric ulcer. In my experience patients with a history of previous ulcers who have later suffered carcinoma have rarely shown the malignancy to be located at the site of previously observed ulcers.

## Hemorrhage

Hemorrhage is the most frequent complication of ulcer. Slight bleeding of course occurs on the surface of all ulcers during the active stage. As little as 30 ml. of ingested blood will produce a black stool and such a stool or a trace of bright blood in the vomitus without general symptoms of hemorrhage is of slight significance. Real massive hemorrhage is usually manifested first by general symptoms such as sudden weakness, vertigo, palpitation, dyspnea, cold extremities and sweating, the symptoms of shock. Frequently such patients are treated for a "heart attack" especially when bright bloody vomitus and/or tarry stools are not noticed by the patient or attendants. I have even seen patients treated for "ptomaine poisoning" because they had arisen in the night and had vomited and passed a diarrheal stool in the dark, the subsequent anemia being attributed to the "poisoning." The real *tarry stool* is not merely black but of a sticky tarry consistency and in severe cases is mixed with liquid dark blood. The *vomitus* may consist of bright blood or clots depending on the length of time it has remained in the stomach. The blood cell count before dilution of the blood by absorption of fluid from the tissues will be the same as before the hemorrhage dropping after twenty-four or forty-eight hours to its minimum if a single hemorrhage has occurred. *Continuous bleeding* is most simply recognized by frequent recording of the blood pressure and pulse, a sustained fall in pressure and increase in pulse rate indicating continuous bleeding. Prerenal azotemia with blood urea nitrogen up to 40 or 60 mg. per 100 ml. reaches its height in twenty-four or forty-eight hours and unless bleeding continues will gradually drop to normal in three or four days. It can therefore also be used as an indication of continued bleeding. In a patient known to have had ulcers previously a hemorrhage may be assumed to be due to ulcer but it must be remembered that massive hemorrhage may be due to blood dyscrasias, ruptured esophageal varices, esophageal, gastric or duodenal neoplasms, severe gastritis and even to vascular lesions in acute bulbar polyomyelitis. Melena alone may also be due to intestinal lesions, inflammatory or neoplastic, to Meckel's diverticulum, to purpura, to hemangioma and to telangiectases. Fortu-



Bread and butter 1 or 2 slices

Pudding custard gelatin ice cream or stewed fruit

*Mid-afternoon*

Same as mid-morning

*Supper*

Same as breakfast or luncheon

*At bedtime and at 2½ hour intervals in night if awake*

Feedings as between meals

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11 Do no more than rule out complications or bleeding from causes other than ulcer such as purpura or varices

5 Order blood coagulation tests, coagulation and bleeding time, prothrombin time, vitamin K determination, and platelet count. Only if coagulation is impaired, prescribe coagulants and vitamin K.

6 Type and match blood for transfusion. No transfusion to be used except for evidence of severe anoxia and air hunger. Then try one or two transfusions of 150 to 200 ml of citrated blood. If no benefit ensues, with the patient almost moribund, I have several times used successfully continuous drip transfusions for thirty-six hours or more of at least 6 to 8 liters of blood. The final cessation of bleeding during continuous transfusions, even though repeated hemorrhages occur at first, may be explained by the process of healing covering the bleeding point.

7 Chart pulse and blood pressure every two hours at first, blood urea nitrogen every two days at last.

8 Test all stools for occult blood until this disappears.

9 Start mineral oil, 1 ounce by mouth every night after second night. Retention oil enema on fourth night and thereafter as required.

10 Do fractional gastric analysis about the ninth day in uncomplicated cases, earlier after mild hemorrhage.

11 Start x-rays the day after the gastric analysis if bleeding has stopped.

12 No gastroscopy until after x-ray examination.

**CONTRAINDICATIONS** It is important to avoid the following: (1) Ice, because externally it increases shock, internally it stimulates gastric circulation. (2) Parenteral fluids, which generally increase blood volume and pressure and cause more bleeding. Rarely, as mentioned above, small transfusions of 150 to 200 ml may be required in severe anoxemia with air hunger. (3) Stimulants (digitalis, epinephrine, and others), because they tend to increase bleeding. Only used in emergency. (4) Alkalies, which stimulate secretion and irritate the bleeding area. (5) Excitement or worry, which increases shock and reaction to which may increase bleeding. (6) Strenuous examinations, manipulations, or treatments to avoid trauma. Do only if absolutely necessary in first few days.

**THE GASTRIC HEMORRHAGE DIET** This should be started as soon as the patient is seen and consists of frequent feedings of liquid nutriment as follows:

#### Formula for Gelatin Milk Feedings

FOOD	AMOUNT	CARBO- HYDRATE	PROTEIN	FAT	CALORIES
Gelatin (powdered)	50 gm		45 gm		180
Dextrose	60 gm	60 gm			240
Cream (20%)	100 cc	3 gm	3 gm	18 gm	180
Milk	900 cc	36 gm	27 gm	27 gm	500
		99 gm	75 gm	45 gm	1150 per L

In making up this formula the milk, dextrose, and cream are first mixed together. The gelatin may be dissolved in a little warmed milk and then added to the mixture. Thus, when cooled, makes a gelatinous mass and must be reheated at each serving. Some prefer to add a rounded teaspoonful of gelatin to each feeding at the time of serving. The drink

nately the same treatment will be adequate for any of these causes except that a ruptured varix might require tamponade, ligation or cauterization

**Treatment** Nature's way of stopping hemorrhage from a bleeding vessel which is the usual cause of ulcer bleeding is by producing shock which enforces rest in a horizontal position reduces blood pressure so as to prevent blowing out of the clot which forms at the site of bleeding and increases the formation of blood coagulation factors. It is important to aid nature in the maintenance of these conditions. The patient should be at rest in bed the blood pressure should not be raised suddenly by intravenous injections of any kind and stimulants should be avoided. There is still considerable controversy about the use of *transfusions*. In most cases there is such a rapid and firm closure at the site of bleeding that transfusions do no harm. But in many cases the blood clot is expelled after transfusions and after each succeeding transfusion further profuse bleeding occurs. Many patients die as a result as shown by statistics which indicate that mortality is doubled with the use of routine transfusion. It is not necessary to encourage blood regeneration since the blood cell counts mount rapidly and spontaneously after hemorrhage. In fact transfusions which provoke further bleeding will wash out the natural coagulant and hematopoietic factors and result in prolonged stubborn anemias. It has recently been shown that transfusion greatly reduces the number of platelets. Avoiding transfusion is particularly advantageous because of the danger of transmission of viral hepatitis. While blood remains in the stomach digestion of the clot in the vessel is prevented by digestion of the blood in the gastric lumen but when this has disappeared the same effect can be secured by *frequent feedings* of soothing liquid foods which combine readily with gastric juice and if possible act as coagulants. Stypsen viper venom plus epinephrine given by stomach tube has been used to control hemorrhage. *Gelatin* accomplishes this end and in addition its principal amino acid glycine is one of the main building stones of the protoporphyrin of hemoglobin.

The following is a copy of the routine treatment for hemorrhage used for many years on my service at the Long Island College Hospital and resulting in a mortality rate of 2 per cent or less as compared with previous mortality rates of 8 to 15 per cent by medical and 4 to 30 per cent by surgical treatments. In our experience age has not been significant patients past fifty years doing as well as younger ones.

#### Routine in Gastric Hemorrhage Treatment

- 1 Order gastric hemorrhage diet
- 2 Treat shock by rest warmth external and sedatives if required
- 3 Quiet apprehension. Reassure patient. Do not isolate.
- 4 Do not take a detailed admission history or make a complete physical examination

used in recent years. Immediate operation usually subtotal resection with only brief preparation by transfusion and attention to electrolyte balance has been so perfected by skillful surgeons that the mortality rate is only slightly higher than for medical care. Studies have shown however that even after operation recurrences are about as frequent as after medical care—so why operate? Delaying operation even for two or three days of observation with transfusions and parenteral fluids to see whether the hemorrhage would stop increases the mortality rate three fold or fourfold and is not desirable. It is best to treat the patient medically as previously outlined. Operation is almost never necessary for bleeding ulcer unless complications such as stenosis or perforation are present. Operating upon all patients past fifty years of age as is done in some places needlessly increases the mortality rate. These patients do just as well as younger ones on medical care as described above.

*Subsequent care* is the same as outlined for uncomplicated ulcer.

### Perforation

Perforation of the ulcer may be of the acute or the so called chronic type.

*Acute Perforation* Acute perforation due to sudden rupture of the ulcer through the peritoneal coat into the free peritoneal cavity usually occurs at the time the ulcer begins. It may therefore occur suddenly without previous ulcer symptoms or with a history of previous attacks over years. When an ulcer has been shown to be present previously the perforation usually can be demonstrated as due to another new ulcer either at the edge of the previous one or some distance away.

The diagnosis is made from the history of the sudden terrific pain followed by more or less profound shock and symptoms of peritonitis. The absence of liver dullness or better the x-ray finding of shifting air in the peritoneal cavity is indicative of perforation of a hollow viscus (See Figs 4-5 p 65). It is usually difficult to rule out perforation of some other hollow viscus acute appendicitis and cholecystitis or acute hemorrhagic pancreatitis. Pancreatitis may be the result of an ulcer perforating into the pancreas. Rarely perforation may be a complication of cancer and appear as its first symptom. Any abdominal calamity may at times be confused with cardiac renal and central nervous system disease.

**TREATMENT** Because of the difficulty of certain diagnosis it is safest to resort to surgical care as soon after a suspected perforation as the patient has recovered from primary shock. Neglect of this precaution is the cause of a high mortality rate. Analgesics should not be given until the diagnosis has been made and operation decided upon for fear of masking the symptoms and signs. The type of operation to be performed is still a subject of dispute among surgeons. Some prefer only closure of the

may be given warmed or cool and is usually sufficiently palatable not to require flavoring. A little vanilla or cocoa may be added if desired.

For the first two or three days after the hemorrhage feed 6 ounces of this mixture every two hours with nothing else by mouth. If asleep the patient should not be disturbed for three or four hours.

On the fourth, fifth and sixth days add to three or four of the feedings one of the following: one egg soft boiled, poached or raw; cereal 3 ounces; custard; Jello or ice cream 3 ounces; and allow water in 3 ounce quantities between feedings.

On the seventh and eighth days add two of the above foods to each of four feedings.

On ninth day order ulcer diet (p. 267).

In patients who are allergic to milk or who object to it a soy bean milk substitute may be used in place of milk and cream or the following formula may be substituted.

#### Formula for Gelatin Water Feedings

FOOD	AMOUNT	CARBO HYDRATE	PROTEIN	CALORIES
Gelatin	50 gm		45 gm	180
Dextrose	90 gm	90 gm		360
Juice of 3 oranges		30 gm		120
Water to 1000 cc				
		<hr/> 120 gm	<hr/> 45 gm	<hr/> 660 per L

This is used the same way as the gelatin milk mixture. In milk allergy subsequent additions consist of only milk free foods.

Vitamin C 0.5 to 1.0 gm and bioflavonoids (vitamin P) 100 mg a day should be ordered with either routine diet.

It will be seen from a study of the foregoing routine that in the case of the gelatin milk feedings a patient taking nine or ten feedings a day will consume nearly 2000 calories in twenty four hours with over 100 gm of protein. In the case of the gelatin water feedings only 1000 calories will be taken with only about 50 gm of protein represented by the gelatin alone or more if the patient can take a protein mixture. All food values increase rapidly as additions are made.

In patients with *stenosis* or *perforation* complicating the hemorrhage the treatment suggested for these conditions must be carried out. In uncomplicated hemorrhage the usual studies are carried out after eight or ten days and complete treatment for ulcer is instituted.

**SURGICAL TREATMENT FOR HEMORRHAGE** Surgery has its advocates. Formerly when surgery was performed only after prolonged medical treatment had failed to stop the bleeding and the patient was in bad shape the mortality rate was so high that most surgeons were afraid to operate. Two new techniques for the surgical approach have been

used in recent years. Immediate operation usually subtotal resection with only brief preparation by transfusion and attention to electrolyte balance has been so perfected by skillful surgeons that the mortality rate is only slightly higher than for medical care. Studies have shown however that even after operation recurrences are about as frequent as after medical care—so why operate? Delaying operation even for two or three days of observation with transfusions and parenteral fluids to see whether the hemorrhage would stop increases the mortality rate three fold or fourfold and is not desirable. It is best to treat the patient medically as previously outlined. Operation is almost never necessary for bleeding ulcer unless complications such as stenosis or perforation are present. Operating upon all patients past fifty years of age as is done in some places needlessly increases the mortality rate. These patients do just as well as younger ones on medical care as described above.

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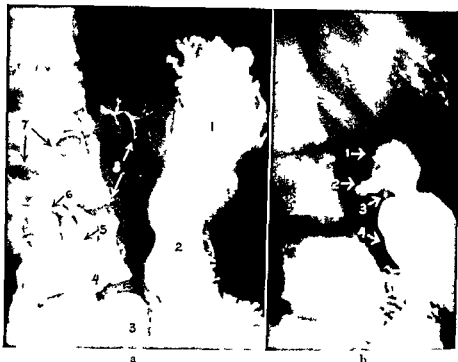
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**Figure 42** Perforated duodenal ulcer *a* Duodeno biliary fistula filled with barium showing (1) fundus (2) corpus (3) antrum (4) cap (5) common duct (6) cystic duct (7) right and left hepatic ducts (8) liver radicles *b* Walled off ulcer lateral view showing (1) cap (2) posterior perforation (3) pylorus (4) antrum

perforation leaving further operation for a future time. Others advise primary gastric resection. In recent years there have been increasing numbers of reports of patients treated without operation; the proponents of this method claiming a lower mortality rate than that of immediate operation. They treat their patients by continuous gastric suction for three or four days with parenteral feedings, antibiotics, and sedation. They claim it is safer than operation in critically ill patients and in those with serious cardiac, renal, or other complications. It is certainly indicated when facilities for operation are unavailable. It is also worthwhile when the patient is first seen without evidence of severe peritonitis two or three days after perforation when presumably the perforation has begun to be walled off.

**Chronic Perforation** This term is often used to describe a slow perforation which has been walled off by the protective response of the omentum in covering up the site of the impending perforation or by adhesions to neighboring organs, most frequently the pancreas but also the gallbladder, liver, or colon, occasionally producing an extravasation or fistula into these organs (Fig. 42).

With the formation of firm adhesions, spill is prevented and an accu-

sory pocket is produced. This pocket whose base is not stomach wall but the tissue of the neighboring organ cannot heal up as in the case of an ordinary ulcer. It may remain open for years producing symptoms either continuous or intermittent often brought on by exertion or trauma. In some such cases there may be bleeding of the chronically inflamed tissues or perforation at the edge of the pocket due to tearing away of adhesions. Rarely cicatricial contraction or granulations in the pocket may cause its obliteration so that the pocket can no longer be seen roentgenographically the symptoms sometimes clearing up entirely. At times the symptoms may persist or cicatrization and contraction may cause stenosis.

These perforated walled off ulcers with accessory pockets causing chronic persistent or frequently recurrent symptoms are often erroneously called "intractable ulcers." They should be recognized at the time of a complete study. X ray films in different positions will show the accessory pocket (Figs 41 *a* and 42 *b*). The probable need for operation should be explained to the patient at the onset of treatment. After suitable preparation consisting in ulcer treatment as previously described a suitable operative procedure is indicated.

### Stenosis

Stenosis may occur (1) as a result of inflammatory reaction induration and edema at the onset of an ulcer (2) as a result of cicatricial contraction following healing of an ulcer (3) from adhesions or cicatricial contractions following perforated or almost perforated ulcers. The stenosis may occur at the pylorus in mid stomach (hourglass stenosis) or in the duodenum. At times a partial stenosis may be made complete by impaction of a swallowed foreign body such as large pits (olive pits) coins or buttons. It is recognized by the cardinal symptoms of "delayed vomiting" the patient vomiting at long intervals all or nearly all the food taken during the interval. When only small amounts of food are being taken the stomach may never become so overloaded as to cause vomiting but the constant epigastric fullness and the belching and eructations of gas or sour material smelling of yeast may indicate the condition. Rapid loss of weight and strength and increasing pallor are also noted. The overnight residue has already been described under Gastric Analysis (p. 260). The x ray finding (Fig. 43) of a large dilated atonic stomach or an hourglass stomach with not only six but often twenty four or even forty eight hour gastric retention of the ingested opaque meal will clinch the diagnosis. Often the ulcer will not be immediately demonstrable. The purpose of treatment should be (1) to give small amounts of concentrated liquid nourishment at frequent intervals to stimulate peristalsis and improve gastric tone (2) daily or twice daily aspiration of all gastric content to prevent overdistention and to act as an index



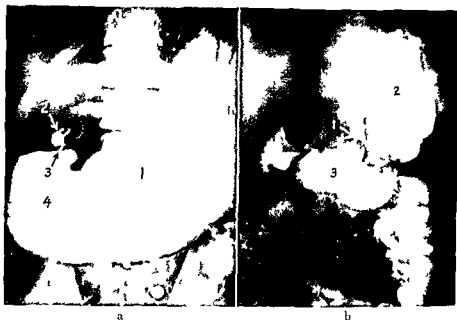


Figure 43 a Stenosis from duodenal ulcer showing (1) stomach dilated (2) posterior perforation causing stenosis (3) pylorus (4) antrum b Hourglass constriction (at arrow 1) showing (2 and 3) upper and lower parts of stomach

of improvement (3) parenteral administration of dextrose and amino acid solutions fortified with vitamins and minerals and (4) any other medication which may be indicated. Transfusions are also of help.

For nourishment I use the same mixture of milk, cream and glucose with added gelatin as for hemorrhage but starting with 4 ounces of the mixture every two hours while the patient is awake and gradually increasing the quantity, adding raw eggs, protein preparations, fruit juices and later cereals, puddings and other foods as indicated by improvement shown in diminishing returns on daily aspiration. In cases in which an inflammatory reaction with spasm was the cause of the stenosis there is a gradual subsidence of the obstruction so that additions to the diet are readily taken. The true organic stenosis may show some improvement but more solid foods, even bread, will not pass the narrow point. Repeated x-ray study after ten days will determine the extent of improvement. Some patients will show complete and permanent relief of the obstruction; others will require operation for relief. In either case general ulcer treatment is indicated in addition (see p. 271).

#### MALIGNANT DEGENERATION

Formerly considered a complication, malignant degeneration has already been discussed. It is recognized today that carcinoma does not occur more frequently in patients who have had ulcers than in the general

population. As has been emphasized, cancer must be ruled out in any case of gastric ulcer because the ulcer may represent ulceration of a cancer.

### POSTOPERATIVE COMPLICATIONS

Postoperative complications in general have been discussed in the general section on the stomach (p. 234). A specific complication or sequel is a *marginal or jejunal ulcer* which resembles other ulcers and is located either along the margin or suture line of the gastroenterostomy or may be entirely in the jejunum. It has been held to be caused by erosion by acid gastric juice. Upon this theory has been based the tendency to resection of more and more of the stomach, even for duodenal ulcer, it being considered necessary to remove all or nearly all the acid-secreting part of the stomach in order to prevent these ulcers. And yet they have continued to occur. Based on the assumption that they are caused by the same factors as the original ulcer, a careful search for neglected focal infections has invariably been rewarded by the finding of such foci in all cases, and their eradication has resulted in permanent healing unless complications had already developed. I am sure it is because I always insist upon removal of all such foci before an elective operation or soon after an emergency operation that I have never had any patient of mine develop such postoperative ulcers unless subsequent infections were neglected.

The *symptoms* may be similar to those of gastric or duodenal ulcer, but may be constant, may be located in the epigastrium, back or elsewhere, and may or may not be accompanied by retrostaltic symptoms, even vomiting. The complications of hemorrhage, perforation and stenosis may also occur and produce similar symptoms.

The *diagnosis* is made by the finding of blood in the gastric contents and by demonstration of deformity and a crater by x-ray film (Fig. 44, a, b).

The *treatment* is the same as for ulcer, consisting of an ulcer diet, attention to focal infections and operation only for complications.

### Complications Requiring Operation

These are stenosis or obstruction by adhesions, free perforation or walled-off perforation and perforation into the transverse colon producing *gastrojejunocolic fistula*.

With *obstruction* there is persistent, often delayed vomiting and the characteristic findings on gastric analysis and x-ray examination. With *free perforation* there are the symptoms of an "acute abdomen." Both require operation.

*Gastrojejunocolic Fistula* After an acute episode of perforation with symptoms subsiding rapidly because the perforation is sealed off by adhesions, this fistula produces, in addition to abdominal pain and dis-

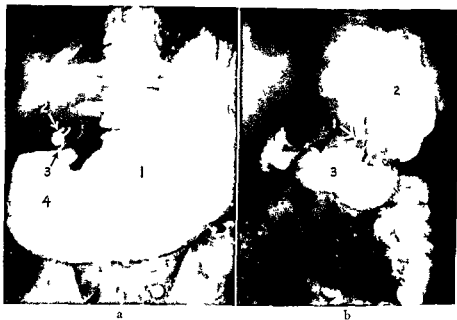


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may appear promptly in the gastric content or vomitus and similar ingested liquids will promptly appear in the feces. Careful x-ray studies by barium meal and enema will also disclose the communication (Fig 44 c). The patient becomes toxic rapidly emaciated dehydrated and anemic and requires careful preoperative care.

Parenteral fluids electrolytes food vitamins and usually blood are indicated. Lavages and enemata are helpful. An early preliminary colostomy proximal to the fistula usually at the cecum or ascending colon permitting expulsion of the highly toxic feces which have been reaching the stomach results in rapid improvement. A few weeks later the more extensive plastic operation can be performed with safety.

**MORE RARE POSTOPERATIVE COMPLICATIONS** These are *gastric mucosal prolapse* through the stoma and *retrograde intussusception* of the jejunum into the stomach. The mucosal prolapse is usually intermittent causing temporary symptoms of obstruction and occasionally bleeding. The intussusception may also occur intermittently and produce periods of obstruction or may cause an acute obstruction with hemorrhage and possible gangrene. The x-ray film and occasionally also the gastroscope will disclose the nature of the obstruction. These complications must be borne in mind in a patient who has had a stomach operation and in whom hemorrhage develops especially if accompanied by obstructive symptoms. The best treatment is surgical.

The so called *dumping syndrome* is discussed in the general chapter on the stomach (p. 235).

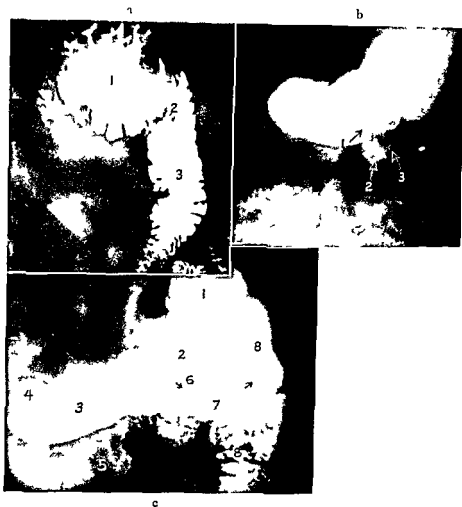
An inexcusable postoperative complication is *gastroileostomy* due to the fact that the surgeon mistook the ileum for the jejunum. The symptoms are similar to those of fistula just described but much more severe. X-ray examination will show the lesion. An extensive operation is required.

## NEOPLASMS OF THE STOMACH

### BENIGN NEOPLASMS

Benign neoplasms of the stomach and duodenum are comparatively rare although with careful x-ray and gastroscopic studies more are being found than formerly. The tumor may be intragastric invading the lumen or extragastric involving the wall and extending outward. Estimates of the percentage of gastric neoplasms which are benign have varied from 1 to 25 per cent. More than half are *epithelial tumors* mostly polypoid and papillary adenomas. Multiple polyps (polyposis) are occasionally encountered. It has been suggested that these tumors may be precursors of carcinoma. The frequent association of gastric polyps with carcinoma elsewhere has been demonstrated.

*Connective tissue tumors* include principally the smooth muscle tumors



*Figure 44* *a* Normal subtotal gastrectomy and gastroenterostomy showing (1) remainder of stomach (2) stoma (3) jejunum *b* Jejunal ulcer (postoperative complications) showing (1) gastrojejunostomy stoma (2) jejunum (3) ulcer crater *c* Gastrojejunocolic fistula showing (1) fundus (2) corpus (3) antrum (4) cap (5) duodenum (6) gastrojejunostomy stoma (7) jejunum (8) colon Arrows indicate direction of flow

tention two groups of characteristic symptoms One is profuse diarrhea the discharges consisting not only of ordinary liquid feces but also sour smelling gastric content demonstrable by the identification of recently ingested food such as milk cereals fruits or vegetables in an almost unchanged form The other consists in nausea fecal odor to the breath and repeated vomiting of feces regurgitated through the fistula into the stomach

**DIAGNOSIS AND TREATMENT** Colored liquids injected into the rectum

### Diagnosis

Aside from obvious bleeding the symptoms are not of much diagnostic help but in unexplained anemia or weight loss with possibly mild digestive symptoms should occasion suspicion. The symptoms and findings of gastritis or ulcer of obstruction of perforation or massive hemorrhage are no different from those due to other causes. It has been mentioned also that age and sex are not distinctive.

### Physical Examination

Physical examination may show nothing abnormal may disclose evidences of concurrent disease or may at times locate a tumor.

### Laboratory Studies

*Fractional gastric analysis* may show no abnormal findings or may show the findings of ulcer or gastritis and will of course disclose blood in the bleeding cases. The persistent finding of occult blood in the stools should occasion a suspicion of neoplasm. If the blood reveals anemia of the secondary type it may be due to hemorrhage of the primary type to destruction of mucosa.

### X-ray Examination

Intragastric and intraduodenal tumors will be seen as defects within the lumen may be so small as to be found only with careful mucosal studies or may be so large as to take up much of the lumen. Single small tumors are most readily missed larger or multiple tumors are more easily identified (Fig. 45). Ulceration may produce defects in the tumor defect so that they may resemble ulcers with induration. Extragastric tumors will cause defects of outline resembling external pressure or malignant involvement. Both kinds of tumors are difficult to differentiate from cancer. A complete gastrointestinal study is indicated especially in view of the frequent association of polyps with cancer elsewhere.

### Instrumental Examinations

*Gastroscopy* is valuable. The tumors can at times be seen when x-ray examination has failed to disclose them and biopsy may reveal the nature of the tumor. *Peritoneoscopy* has been used to determine the nature of the extragastric tumors.

### Treatment

When a tumor has been demonstrated to be present its surgical removal is imperative because of the possibility that the tumor is malignant or may become so. The type of operation depends on the size and location of the tumor but radical resection is to be preferred. The patient

the usually small leiomyomas being the most common. Hemangiomas and telangiectases are rare as are fibromas. Hamartomas have been found. Neurofibromas extremely rare may be associated with generalized neurofibromatosis (von Recklinghausen's disease). Myomas, lipomas and dermoid cysts have also been seen. It has been suggested but not proved that malignant degeneration may take place in some of these tumors. *leiomyosarcoma*, *neurosarcoma* and *fibrosarcoma* being quoted as possible examples.

Boeck's sarcoid which in the stomach resembles a chronic granulomatous gastritis is seen in tuberculosis but distinguished from it by the finding of Schaumann inclusion bodies may be classed among the tumors.

Eosinophilic granuloma is a rare cause of a prepyloric tumor resembling cancer. It may be in accompaniment of eosinophilic leukemia.

Aberrant pancreatic tissue in the stomach and crop may also be included among tumors.

There is always more or less accompanying gastritis and duodenitis which may be the cause or the result of the presence of the tumors. Necrosis with ulceration or bleeding may occur. Perforation is unusual. Some tumors may remain small others may grow rapidly to enormous size. Some become calcified.

### Incidence

All these tumors may occur at any age although they are more common in middle age or later. They occur equally in both sexes.

### Etiology

Little is known about the cause of benign tumors. Some are believed to be congenital, some are hereditary and some the result of irritation mechanical or inflammatory.

### Symptoms

There are no characteristic symptoms. There may be no symptoms at all or there may be retrostaltic symptoms from irritation. Typical peptic ulcer symptoms may occur. If located at a narrow point such as the cardiac orifice, the pylorus or the duodenum they may cause more or less severe obstructive symptoms. If more or less pedunculated and in the prepyloric region they may be carried through the pylorus pulling the gastric mucosa after them and producing narrowing or even obstruction in the duodenum due to the intussusception. The mucosa over the tumor may ulcerate and bleed producing secondary anemia. If ulceration is extensive and blood vessels are injured massive hemorrhage may be the first or most prominent symptom. In multiple polyposis melanin spots on the lips and buccal mucosa or on the fingers and toes have been observed.

a rounded ulcer in the mucosal surface such a lesion called ulcer carcinoma may defy differentiation from a gastric ulcer with induration. Intramural cancer is rare. Carcinoma has also been found in the margin of a gastrojejunal stoma. Rarely the stomach or duodenum becomes involved secondarily from carcinoma of the pancreas, biliary tract, transverse colon (into which perforation produces a gastrocolic fistula) or even from renal or splenic tumors. Squamous cell carcinoma may rarely spread from the esophagus to the gastric fundus. Carcinoid tumors now considered malignant are extremely rare in the stomach and duodenum. Of the malignant lymphomas, lymphosarcoma and Hodgkin's disease usually involve only the deeper wall of the stomach but ulcers frequently develop in the overlying mucosa. Sarcoma may originate in a leiomyoma. Reticulum cell and neurogenic sarcomas and melanomas are rare.

Metastases to the liver and regional lymph nodes are most frequent and may be extensive even when the primary gastric lesion is small. Occasionally adenocarcinoma may extend upward into the esophagus for considerable distances. It may also spread late to the diaphragm, mediastinum, lung, heart and brain. Involvement of the left supraclavicular lymph node known as "Virchow's gland" indicates extensive spread and incurability.

### Etiology

Etiology has been discussed under the general subject of gastrointestinal cancer (p. 120). The question of heredity as a factor may some day be determined when more states adopt compulsory registration of cancer cases as in Connecticut. Heredity appears to play an important role. Atrophy of the gastric mucosa (chronic gastritis) with achlorhydria and pernicious anemia may be predisposing factors or may be due to a common factor causing these changes as well as the cancer. Gastric polyps and gastritis polyposa have been suggested as precursors but they are more rare than cancer. Ulcers have been shown not to undergo malignant changes any more than normal tissues. Prolonged ingestion of irritants such as overheated fats, food dyes, strong spices, condiments and alcohol does not appear to show a constant relationship although a history of habitual ingestion of very hot liquids, especially hot water on arising, is frequently obtained. Miners exposed to iron dust for ten or more years are prone to suffer stomach cancer. Chronic irritation from foods to which a stomach is allergic and the ingestion of carcinogenic substances are also to be considered causes. Increasing evidence of a viral etiology of cancer is accumulating.

### Diagnosis

Diagnosis is useless unless made early enough to permit cure. In the knowledge that many early cases are overlooked, universal screening has been recommended but is not practicable. A thorough and complete



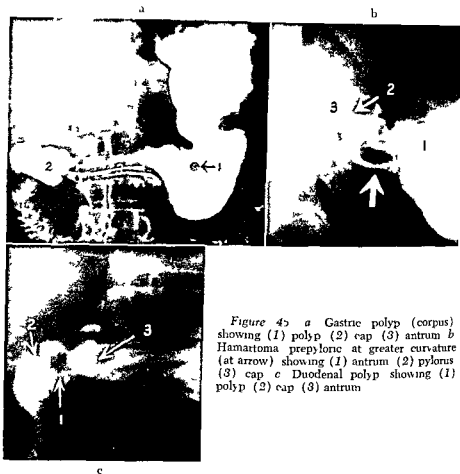


Figure 4: a Gastric polyp (corpus) showing (1) polyp (2) cap (3) antrum b Hamartoma prepyloric at greater curvature (at arrow) showing (1) antrum (2) pylorus (3) cap c Duodenal polyp showing (1) polyp (2) cap (3) antrum

should be carefully prepared for operation and treated postoperatively as has been described

#### MALIGNANT NEOPLASMS

The stomach is the most frequent site of cancer in the male and stands fourth in frequency in the female in whom breast uterine and intestinal cancer are more frequent in the order named. About 30 000 persons die of gastric cancer each year in this country. Duodenal cancer is extremely rare and is usually secondary. (See Gastrointestinal Cancer page 119)

#### Pathology

Primary adenocarcinoma is the usual type found in the stomach but colloid medullary papillary and scirrhous (fibrocarcinoma) varieties occur. The last named starts as a small localized area near the pylorus eventually involving the whole stomach and is then called linitis plastica or "leather bottle stomach". Occasionally a localized area of such a fibrocarcinoma on the posterior wall or lesser curvature may develop

a rounded ulcer in the mucosal surface such a lesion called ulcer carcinomatosus may defy differentiation from a gastric ulcer with induration. Intramural cancer is rare. Carcinoma has also been found in the margin of a gastroduodenal stoma. Rarely the stomach or duodenum becomes involved secondarily from carcinoma of the pancreas, biliary tract, transverse colon (into which perforation produces a gastrocolic fistula) or even from renal or splenic tumors. Squamous cell carcinoma may rarely spread from the esophagus to the gastric fundus. Carcinoid tumors now considered malignant are extremely rare in the stomach and duodenum. Of the malignant lymphomas, lymphosarcoma and Hodgkin's disease usually involve only the deeper wall of the stomach but ulcers frequently develop in the overlying mucosa. Sarcoma may originate in a leiomyoma. Reticulum cell and neurogenic sarcomas and melanomas are rare.

*Metastases* to the liver and regional lymph nodes are most frequent and may be extensive even when the primary gastric lesion is small. Occasionally adenocarcinoma may extend upward into the esophagus for considerable distances. It may also spread late to the diaphragm, mediastinum, lung, heart and brain. Involvement of the left supraclavicular lymph node known as "Virchow's gland" indicates extensive spread and incurability.

### Etiology

Etiology has been discussed under the general subject of gastrointestinal cancer (p. 120). The question of heredity as a factor may some day be determined when more states adopt compulsory registration of cancer cases as in Connecticut. Heredity appears to play an important role. Atrophy of the gastric mucosa (chronic gastritis) with achlorhydria and pernicious anemia may be predisposing factors or may be due to a common factor causing these changes as well as the cancer. Gastric polyps and gastritis polyposa have been suggested as precursors but they are more rare than cancer. Ulcers have been shown not to undergo malignant changes any more than normal tissues. Prolonged ingestion of irritants such as overheated fats, food dyes, strong spices, condiments and alcohol does not appear to show a constant relationship although a history of habitual ingestion of very hot liquids, especially hot water on arising, is frequently obtained. Miners exposed to iron dust for ten or more years are prone to suffer stomach cancer. Chronic irritation from foods to which a stomach is allergic and the ingestion of carcinogenic substances are also to be considered causes. Increasing evidence of a viral etiology of cancer is accumulating.

### Diagnosis

Diagnosis is useless unless made early enough to permit cure. In the knowledge that many early cases are overlooked, universal screening has been recommended but is not practicable. A thorough and complete

gastrointestinal study should be made in all patients with pernicious anemia or complaining of recent persistent gastrointestinal symptoms especially in those having a family history of cancer. Only in this way will early cases be discovered.

### Symptoms

Unfortunately many patients do not have any early symptoms or they are so slight as to be overlooked. Occurring in late middle age they are often attributed merely to getting old. Mild retrostaltic symptoms such as epigastric fullness or gas after meals, belching, sour eructations or anorexia if occurring in a person at any age but especially after age forty without any previous gastrointestinal symptoms are very suspicious of early cancer. Carcinomatous ulcer may produce typical symptoms of a simple ulcer and if these occur without a history of previous similar attacks a cancer is also to be suspected. A carcinoma high up in the fundus may cause no symptoms or at times only mild symptoms due to secondary cardiospasm long before it has invaded the cardiac orifice. As the lesion becomes more advanced the anorexia especially a distaste for meat becomes more marked. With increasing food intake weight loss, anemia and constipation follow. Later complications such as stenosis due to the size of the growth and hemorrhage or perforation may occur. At times the patient will have no appreciable symptoms until metastases produce liver enlargement, pain or enlarged neck glands. Thrombophlebitis in the extremities is occasionally seen before symptoms of cancer occur.

### Physical Examination

When the cancer is still small examination will be entirely negative. Pallor, evidences of loss of weight, tenderness, a mass or enlarged lymph nodes (Virchow's gland) often mentioned in the textbooks are evidences of an advanced lesion usually incurable. Rarely can a patient with a palpable mass be expected to show a resectable lesion and of course distant enlarged lymph nodes proved by biopsy to be malignant make the prognosis hopeless. Examination in such cases must therefore be confined to an evaluation of the patient's general status as an operative risk and should include such laboratory studies as blood cell counts, chemistries and serology, renal and hepatic function tests, electrocardiography and other indicated procedures.

### Laboratory Examinations

Laboratory examinations of value in making a diagnosis are available but none are consistently specific. Unfortunately until a specific biologic or biochemical test for cancer is developed or until tracer studies with radioisotopes or labeled metabolites or immune bodies are further de-

veloped we must use the means at hand to make a diagnosis as early as possible. Four procedures are of real value viz fractional gastric analysis, exfoliative cytology, gastroscopy and x-ray study. The last named is of the greatest single value although enhanced by combination with one or more of the others.

Fractional gastric analysis is of distinct value. In intelligent patients not more than 1 or 2 per cent will refuse to swallow the tube even repeatedly, and even among clinic patients not more than 5 per cent refuse it. The skillful passage of the Relfuss tube by mouth should not occasion enough trauma to cause any bleeding unless an ulcerative lesion is present. If blood is obtained on gentle suction before the tip has entered the stomach, an esophageal lesion is to be suspected. If visible blood is found in the fasting stomach indicating an ulcerative lesion a cancer must be ruled out. If bleeding continues after the stomach contents have been completely aspirated and a gentle lavage has been done, it is doubly significant. The degree of acidity in the gastric contents cannot be considered of definite diagnostic value. Although hypochlorhydria or achlorhydria is found in 70 per cent or more of gastric cancers, some of the highest acidities I have ever seen were found in the gastric contents of patients with even large inoperable cancers. The old emphasis on the presence of lactic acid as an indication of cancer based on the theory that the cancer tissue produces lactic acid is being re-evaluated. It is generally conceded, however, that the lactic acid is due to fermentation in stagnant gastric contents by *Bacillus acidilactici* which will not grow in the presence of free hydrochloric acid and will therefore occur only in achlorhydric stomachs with pyloric stenosis (Fig. 46).

Cytologic study of the fasting content and stomach washings by the Papanicolaou method may disclose definite cancer cells and be of great help in diagnosis even before a growth has become visible. This cytologic study has been further improved by the use of various techniques for

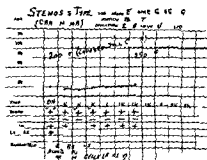


Figure 46. Fractional gastric analysis—stenosis from cancer. Secretion no free acid, motility overnight retention, large residue at end, admixtures blood in all specimens. In fasting contents food remnants, lactic acid, lactic acid bacilli and carcinoma cells (rarely).

obtaining specimens. The best is an inflatable abrasive balloon at the tip of a stomach tube soft silk fibers enmeshing cancer cells of even very early lesions. Gastric suction biopsy under x-ray guidance may be of value. The apparatus for performing this test permits a small piece of mucosa to be sucked through a small hole in a cylinder and then cut off by a small knife blade in the cylinder.

*Gastroscopy* is more disagreeable to the patient but with a proper psychologic approach and with careful anesthetization of the throat causes surprisingly little discomfort and most patients do not refuse repeated examinations. Unfortunately the area of gastric mucosa visualized is limited and often suspected areas cannot be seen. When such lesions can be seen the new Benedict operating gastroscope permits the removal of specimens for biopsy and in such cases the diagnosis can often be fairly well established by this method. Through the *esophagoscope* lesions of the cardiac end of the stomach may at times be seen and biopsies obtained.

*Electrogastrography* similar to electrocardiography is a new method of examination. Study of electrogastrograms may some day be of value.

*X-ray Examination* X-ray study made by a competent roentgenologist is of the greatest value in making a diagnosis. *Fluoroscopy* alone if done by a tyro, is of no value whatever. In the hands of the most expert fluoroscopist it is of definite although limited value. However by moving the patient about by palpation in upright and recumbent positions adhesions to and compression from neighboring organs may be demonstrated. At times small areas of stiffening of the gastric outline may be seen indicating induration. A stiff wide open pylorus with rapid passage of gastric content through it is suggestive especially if there is gastric retention in six hour films. At times real defects often with craters indicating ulceration can be discovered. Small polypoid areas may be disclosed. Cardiospasm may be demonstrated and should lead to a suspicion of fundal involvement. However even large inoperable lesions may be entirely overlooked. Combined with spot films the value of fluoroscopy is much enhanced but even with such a help it is an uncertain quantity.

*Cinefluorography* the taking of frequent pictures as seen on the fluoroscopic screen is being constantly improved and may be of help in screening for early unsuspected cancer.

*Roentgenography* constitutes the only really dependable method of diagnosis in early gastric cancer. It must include a number at least six to twelve films in the prone position and several in both oblique and lateral positions and in the supine position. Films in the erect and semi-recumbent position are also necessary. There is no advantage in describing here the constant irregular defects the large craters with irregular bases the general decrease in the lumen the prepyloric annular defect

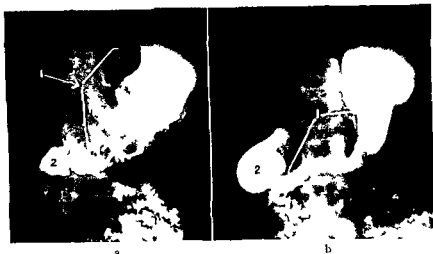


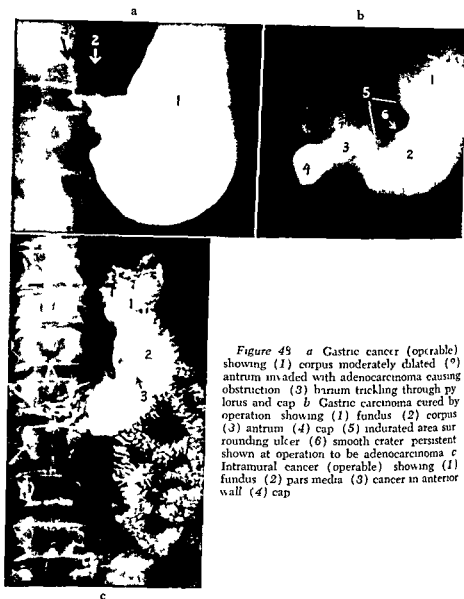
Figure 4— a Gastric cancer extensive showing (1) carcinoma in olving stomach from cardia to pylorus (2) diodenal cap intact b Scirrhus carcinoma (linitis plastica) from fundus to pylorus (1) showing (—) duodenal cap intact

or the fairly atonic stomach with hypertrophied wall seen in partial pyloric stenosis due to cancer. Those are the findings of advanced cancer probably inoperable (Fig. 47)

### Early Diagnosis

To make a sufficiently early diagnosis it is necessary to discover the small indurated areas, defects or polypoid changes which may occur in any part of the stomach but have been said to occur mainly in the pyloric antrum. Or the cancer may appear to be an indurated ulcer with a smooth crater and surrounding induration occurring usually in the antral region along the lesser curvature or posterior wall (Fig. 48). Any such area if resected by the surgeon may show only equivocal changes in the microscopic sections on which the pathologists will disagree in regard to the diagnosis. No one will know whether the patient had a cancer and the prognosis will be in doubt. The only real way to diagnose in a doubtful case is to repeat the x-ray examination in three or four weeks. If all traces of the lesion have then disappeared its benign character will be established, whereas persistence or increase in the size of the indicated lesion will pretty conclusively call for surgical extirpation. In the case of suspected carcinomatous ulcer it is well to be sure that decrease in size of the crater is not due to its being filled in with cancer tissue. In such a case, however, the base of the crater or the area previously occupied by the crater is always somewhat irregular in outline and the surrounding induration can be demonstrated to have spread.

A lesion on the *greater curvature* can safely be called cancer because



*Figure 49 a* Gastric cancer (operable) showing (1) corpus moderately dilated (2) antrum invaded with adenocarcinoma causing obstruction (3) barium trickling through pylorus and cap *b* Gastric carcinoma cured by operation showing (1) fundus (2) corpus (3) antrum (4) cap (5) indurated area surrounding ulcer (6) smooth crater persistent shown at operation to be adenocarcinoma *c* Intramural cancer (operable) showing (1) fundus (2) pars media (3) cancer in anterior wall (4) cap

benign ulcer rarely if ever occurs there Tumors of the fundus can usually be demonstrated with the patient in the supine position When the fundus fills uncertain findings are often clarified by films in oblique positions or by mucosal studies aided by air inflation or by the use of effervescent powders Involvement of or spread into or from neighboring organs can often be shown Figure 49 *a* shows a greater curvature cancer which has involved and perforated into the upper descending colon Cancer of the duodenum usually secondary shows in irregular defect

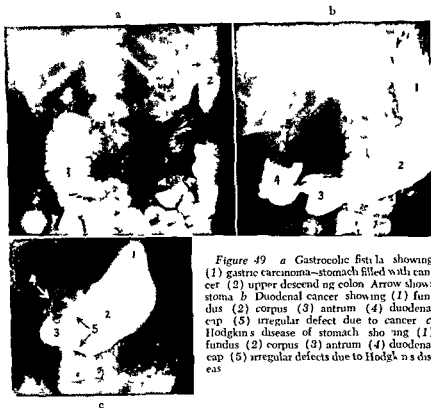


Figure 49 *a* Gastrocolic fistula showing (1) gastric carcinoma—stomach filled with cancer (2) upper descending colon Arrow shows stoma *b* Duodenal cancer showing (1) fundus (2) corpus (3) antrum (4) duodenal cap (5) irregular defect due to cancer *c* Hodgkin's disease of stomach showing (1) fundus (2) corpus (3) antrum (4) duodenal cap (5) irregular defects due to Hodgkin's disease

as in Figure 49 *b* Cancer of the margin of a gastroenterostomy resembles a marginal ulcer Lymphomas may show only stiffening or irregularity of the gastric outlines (Fig 49 *c*)

Benign neoplasms are difficult to differentiate in many cases Gastric syphilis resembles cancer radiographically as do also tuberculosis actinomycosis anthrax and amyloid disease Benign or congenital pyloric muscle hypertrophy may occasion confusion A complete gastrointestinal series barium enema study and cholecystograms should not be neglected because they may be of importance in demonstrating such involvements

### Treatment

*Prophylactic* treatment should consist in general in the avoidance of gastric irritation from ingestion of the substances mentioned under Etiology (p 283) adequate treatment of any diseases which might cause gastric irritation and prompt removal of benign neoplasms especially polyps Repeated gastrointestinal studies at middle age and later are of obvious value

*Radiotherapy* especially when combined with the administration of



chemicals such as nitrogen mustard has been found moderately useful in checking some lymphomatous growths and even curing a few but there is no successful treatment for gastric carcinoma except thorough extirpation. Radiotherapy may at times cause some temporary decrease in the size of a gastric cancer but the side effects of the treatment are so distressing to the patient that this form of palliative treatment is not justified. The use of radiotherapy before and after operation formerly much in vogue as a prophylactic measure to destroy possible metastases has not been of sufficient value to subject the patient to the disagreeable effects except in the case of lymphoma in which it is of distinct value. Reticulum cell sarcoma may be destroyed at times by radioactive phosphorus.

*Surgical Treatment* Until some constitutional treatment for cancer is discovered surgical extirpation of the primary lesion and of all extensions and metastases remains the only hope of cure. This hope is more and more dissipated as the cancer continues to grow. Yet on rare occasions even in fairly far advanced cancer when metastases are fortunately within reach of the surgeon's knife cures have been occasionally reported. It is therefore always worth while to try surgery in any case not actually in the terminal stage or not showing distant metastases such as peritoneal implants, lymphatic metastases at the porta hepatis or behind the head of the pancreas or of course a Virchow's gland. The tendency is toward more and more radical surgery with total gastrectomies frequently performed even by hospital residents and extensive resections of the stomach and involved neighboring organs occasionally proving successful. In localized lesions however the high mortality rate and bad sequelae of total gastrectomies are resulting in less frequent recourse to this procedure except when the cardiac end of the stomach is involved. Even in cardiac cancers partial proximal gastrectomy has been successful. Even if the extensive operations result in less than 5 per cent cures they are preferable to inaction with 100 per cent mortality. Hormonal therapy does little more than relieve symptoms.

*Palliative surgery* is justifiable in patients with even extensive spread of metastases or with pyloric obstruction due to inoperable growth. Simple gastroenterostomy if it can be done may give a patient six months or more of comparative comfort. A gastrostomy for an obstructive growth at the cardia or a jejunostomy for antral involvement may prolong life for a while. The new spool like prosthesis made of nylon invented by Barnes may be sewed into a gastrostomy or jejunostomy with excellent results. It prevents the leakage with digestion and corrosion of the skin about the stoma which caused so much pain and suffering in the past. Liquid balanced feedings as mentioned elsewhere (p. 271) can keep the patient nourished and comfortable.

*Preoperative medical care* is of the utmost importance and should be

in the hands of the internist. Too often we see a patient on whom a diagnosis of gastric cancer has been made rushed into the hospital for immediate operation as if a delay of even a few days would endanger the patient. On the contrary, except when perforation has occurred, adequate preparation for even as long as a few weeks or a month may spell the difference between life and death for the patient. Even during the period of three or four weeks recommended for a repeated radiographic study to determine the accuracy of a cancer diagnosis, adequate dietetic and general care surely will improve the patient's chances of recovering from an operation if it is indicated. In early cancer without complications and with the patient in good general condition, a shorter time may suffice during which the patient should rest but should not be confined to bed because of the danger of lung congestion. He should receive a nutritious well balanced diet such as recommended for ulcer (p. 267) consisting of frequent feedings and plenty of fluids. Water and electrolyte balance should be established. One or more transfusions may be indicated.

In the patients in whom complications have developed these will require special attention. In cases with lower esophageal or pyloric stenosis it is well to lavage once or twice daily and to give feedings every two or three hours of one half to one glass of a mixture of milk, cream, glucose and gelatin (Gelatin Milk Mixture, p. 271). Two or three raw eggs and amino acid or protein distylate preparations should be added to this mixture every day and concentrated vitamins and minerals should be administered orally or parenterally. Parenteral feedings and transfusions will usually be required. In cases with *gastrocolic fistulas* a preliminary colostomy proximal to the fistula or an ileosigmoidostomy, done ten days or two weeks before the more extensive operation is attempted, will permit feedings and will often transform a moribund patient into one who can easily survive very radical surgery. In cases in which the radical operation is found to be contraindicated, such an operation may give the patient a few months of comfort with gain in weight and strength and freedom for some time from the distressing gastrocolic diarrhea and fecal vomiting.

*Psychologic care* of the gastric cancer patient is also of great importance. Whether operation appeared to give promise of cure, whether a purely palliative operation was performed, or whether at exploration such extensive involvement was found that nothing could be done, I am firmly of the opinion that only in exceptional cases, when hardships might be caused to others, is it unwise to let the patient know the diagnosis or prognosis. It is important, however, that some member of the family should always be informed. In a case in which nothing could be done at operation, I explain to the patient that the operation was a difficult one, that the full benefit from the procedure will not become apparent for many months, and that during the interim a restricted but nutritious

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hours Within two or three days additions should be made starting with cereals custards puddings soft eggs milk toast or ice cream gradually increasing the size of the feedings until after six or seven days the patient is on an ulcer diet Postoperative care is fully described in the section on The Stomach (p 232) the ulcer diet under Gastric and Duodenal Ulcer (p 267)

Even though only a small part of the stomach remains or the stomach has been entirely resected and food is entering the jejunum directly a smooth diet such as the foregoing will usually be well tolerated although the quantities will generally need to be small at first Gradually vegetables fruits and later meats should be added to the diet until in a few months the patient is on a fairly full regimen On early feedings with gradual additions the so-called dumping syndrome is rarely if ever encountered

It is surprising how much a patient can eat when most or all of the stomach has been resected It is important and often difficult to have postoperative cases observed at regular intervals to have careful blood determinations to establish whether loss of the stomach and its intrinsic factor may require treatment for pernicious anemia and to have periodic x ray studies made In cases of local recurrence of cancer an attempt at further resection is indicated not a palliative jejunostomy

### Summary

In gastric cancer the patient's survival depends upon early diagnosis and early operation Early diagnosis is based on suspicious early symptoms and careful x ray and laboratory diagnosis Preoperative and post operative medical care contribute greatly to the success of operation Incurable cases can be greatly helped by adequate medical attention including diet medication and psychologic management

## GENERAL DISEASES AFFECTING THE STOMACH

### ACUTE DISEASES

It is to be expected that the stomach should be affected by general diseases It is undoubtedly true that in the course of various acute generalized infections such as pneumonia virus infections and typhoid fever the gastric mucosa will show some changes usually not enough to be called gastritis but often sufficient to give some more or less mild gastric symptoms These are usually relieved by giving the patient a bland but well balanced diet with added vitamins and with adequate water in addition to the measures necessary to combat the general infection

### CHRONIC DISEASES

The chronic diseases causing gastric irritation include metabolic endocrine biliary tract, renal pelvic and other diseases These also require

diet will be necessary together with proper medication. I try to predict the time when improvement will occur as that when the patient can be expected to die. In the case of the palliative operation the patient should be warned that too rapid additions to the diet may occasion recurrences of symptoms. Later when such symptoms occur as they are bound to the patient is prepared for a return to the liquid feedings and increased medication.

In the case in which there is such definite evidence of inoperability with perhaps distant lymph node involvement or liver or other metastases that no operation is attempted it is well to tell the patient that he has a peptic ulcer which will take a long time to heal under careful dieting and medication. The diet will depend upon the extent of involvement. If obstruction has occurred at the cardia or pylorus or if the stomach has been much reduced in size a well balanced liquid diet such as recommended above may be necessary to prevent vomiting or regurgitation and occasional lavage will be required. Protein hydrolysates or amino acid mixtures by mouth or parenterally will provide for additional protein and fruit juices and vitamin supplements are of value. In some inoperable cases I have observed remarkable improvement and gain in weight and strength for a while on such a regimen. Instances have been reported of patients living up to four or six years after being declared inoperable.

In the *incurable patient* vomiting may often be controllable by such agents as Dramamine or Thorazine but later when it becomes intractable will require narcotics. Pain may at first be relieved by the diet. Later it may be somewhat alleviated by aspirin, coal tar analgesics or codeine combined with tranquilizers, barbiturates, chloral or other hypnotics. Finally opiates should be used freely in increasing doses with the knowledge that the patient will become an addict. When the patient becomes unable to swallow parenteral or rectal administration may become necessary. Little benefit can be expected from snake venom and other medications supposed to relieve intractable pain. Nerve blocking or cordotomy is rarely justifiable. Androgenic or estrogenic therapy is seldom of any use.

To the layman the story of prolonged and severe suffering of a cancer case is potent reason for avoiding a study which might reveal a cancer. In inoperable cases the family doctor should not simply tell the family that nothing can be done asking them to send for him when the patient is obviously dying as is often thoughtlessly advised but should take active charge of the case easing the patient's symptoms and comforting the family.

*Postoperative care* in the patient who has had a more or less extensive resection of the stomach should consist not only of transfusions and parenteral fluids and feedings but also liquid oral feedings started as soon after operation as the patient can swallow often within five or six

secretion Today with simple and adequate therapy for syphilis tertiary lesions should rarely be encountered

### Gastric Tuberculosis

Tuberculosis is in almost the same position as syphilis. It is a lesion of advanced tuberculosis and is rarely seen. Here also the symptoms and findings may closely resemble those of cancer and it is difficult to differentiate them without operation. The modern therapy of tuberculosis is beneficial in gastrointestinal and peritoneal tuberculosis. In advanced pulmonary tuberculosis with laryngeal involvement vomiting may be the most prominent symptom even though the stomach is not involved.

### Leukemia

In leukemia the stomach rarely may show lesions resembling malignancy. It usually can be recognized by the blood changes.

*Pseudoleukemia* shows a hyperplasia of lymphoid tissue which is difficult to differentiate from a malignant lymphoma except by biopsy.

### Mycotic Diseases

*Actinomyces* as well as other fungi have been described as rarely invading the stomach. Finding the organisms in gastric contents on culture by serological agglutination and skin tests, blood cultures or animal inoculation seems to indicate that they may be causing the gastric disturbance. The symptoms and findings may resemble those of any gastric disease even cancer. In bizarre cases not responding to ordinary therapy and with a history of possible mycotic infection (p 623) a study for this as a cause may be rewarding.

### Collagen Diseases

*Scleroderma* although primarily a skin disease but occurring also at times in other organs has been much more rarely seen in the stomach than in the esophagus. In the stomach it resembles cancer. It should be suspected if present in the skin in a patient whose x ray films resemble those of cancer. *Polyarteritis nodosa* (see p 99) may resemble ulcer. A gastroscopic biopsy might confirm the diagnosis.

### Vascular Diseases

Vascular diseases may produce gastric lesions. The arteriosclerotic ulcer has been mentioned. Varicosities secondary to portal hypertension are occasionally seen as an extension of esophageal varices and may account for hemorrhage. Abdominal aneurysm also mentioned elsewhere (see p 627) may rupture into the stomach or duodenum causing sudden death.



Figure 50 Gastric cancer ulcerating in a syphilitic showing (1) large ulcer crater (2) cicatricial narrowing often considered characteristic of syphilis

careful attention to diet. *Chronic infectious diseases* especially syphilis and tuberculosis formerly of considerable importance are so rare today as to require only passing mention.

### Gastric Syphilis

In the *secondary stage* syphilis usually shows mucous patches and hyperemia of the gastric mucosa which have been recognized by gastroscopy. In the *tertiary stage* gummas and areas of induration often with ulceration and bleeding were formerly described and were difficult to differentiate from cancer or ulcer. With the recent increase in neglected venereal diseases we may expect to see more gastric syphilis. A positive serological examination does not necessarily indicate that the gastric lesion is syphilitic since cancer occurs just as frequently in syphilitics as in normal persons (Fig 50). Disappearance or scarring of an area resembling cancer when the patient is under intensive antisymphilitic treatment has usually been considered evidence of its syphilitic nature. However gastric ulcers will heal spontaneously in a syphilitic as well as in normal persons. In *tabes dorsalis* the gastric crises although really not gastric but so named because of the location of the girdle pains have at times been shown to be accompanied by an achylia or again by hyper

*Gastric neuroses* have been much exploited. I have seen patients with large palpable and in one case easily visible gastric cancer who had just finished courses of psychiatric care including shock treatments for so called gastric neuroses. Symptoms not clearly understood because of insufficient study are easily attributed to "nerves." Frequently patients are told by their doctors that they have "nervous stomachs." Even peptic ulcer has been included among psychogenic diseases. That this is not so is easily demonstrable as has been discussed under Peptic Ulcer. In my experience any nervous symptoms associated with ulcer are due to the repeated occurrences of ulcers and are entirely alleviated when the patient realizes that no more ulcers are occurring. When real psychiatric conditions are present it is of course necessary to have the patients treated by a psychiatrist but any patient with gastrointestinal symptoms should have a complete gastrointestinal study at the same time.

#### FOREIGN BODIES

Foreign bodies of various kinds which failed to become lodged in the esophagus have been found in the stomach. These include (1) buttons coins pits bones pins tooth picks matches small toys dentures and broken-off teeth accidentally swallowed some by children but many by adults (2) objects accidentally swallowed by people in whose occupations tacks nails pins needles buttons and other articles are held in the mouth during work (3) objects swallowed by side show exhibitors such as broken glass or parts of larger objects which have been broken off or lost (4) In hospitals for mental disease it is not unusual for surgeons to remove repeatedly from the stomachs of inmates knives forks

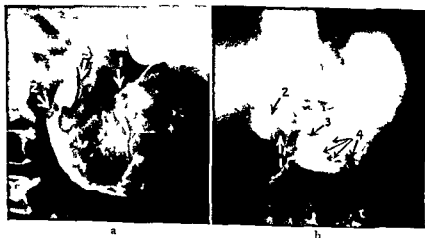


Figure 51 a Phytobezoar due to persimmons showing (1) phytobezoar (2) pylorus (3) cap b Phytobezoar made complete by olive pits showing (1) narrow pylorus (2) faint duodenal cap (3 and 4) many olive pits



**DISEASES OF THE NERVOUS SYSTEM AFFECTING THE STOMACH**

Diseases of the nervous system may affect the stomach usually through the sympathetic and parasympathetic systems which exert a controlling influence on gastrointestinal function. Spasms and motility disturbances of the stomach may occur as a result of disease elsewhere as in the chest or pelvis or from operations and scars. *Tuberculosis dorsalis*, multiple sclerosis and spinal cord tumors may affect both secretion and motility producing vague gastric symptoms even nausea and vomiting. The implication that the diencephalon may be concerned with the formation of ulcer has never been proved. The frontal lobe and especially the prefrontal cortex have also been considered a cause of ulcer. Transection of the vagus nerve and operations upon the prefrontal area have not had any permanent effect on ulcers. A complete gastrointestinal study should be performed because organic disease may be present in addition to the nervous trouble.

**EMOTIONAL DISTURBANCES**

Emotional disturbances have long been known to have some effect on the stomach. Anger, grief, worry, fear, jealousy and embarrassment may cause sensations varying from anorexia to actual pain. However, though these influences cause symptoms of short duration, the gastrointestinal tract soon adapts itself to them and goes on normally. An exception to this is when conditions are all set for the occurrence of an ulcer or an ulcerative colitis, requiring only the trigger to set off an attack. However, when all causes of ulcer have been removed and when all allergens causing ulcerative colitis are being avoided, the same emotional influences will not produce these diseases.

*Anorexia nervosa*, a periodic or chronic loss of appetite, is a common occurrence in mental and endocrine diseases. Such patients may refuse food to the point of emaciation and serious metabolic disturbances. I have obtained little if any help from psychiatric care of such patients. Forced feedings, if necessary by nasal tube at the start, will usually correct the condition. It is important to make a complete gastrointestinal study in order to rule out organic disease.

*Bulimia*, or the desire to overeat, may be psychogenic but may also be due to increased metabolism as in hyperthyroidism, to hypoglycemia as in diabetes, or to organic disease as in ulcer. It is therefore also important to rule out all other causes by a careful study before deciding that bulimia represents an increased craving for love or an aggressive tendency to grab or possess.

*Nervous vomiting*, nervous dysphagia and, in the opinion of psychiatrists, even cardiospasm have been attributed to fears, frustrations and obsessions. If organic causes have been ruled out, psychiatric care may relieve these symptoms.

the pylorus may go down the intestinal tract and be expelled or may become lodged at some narrow point. Some may perforate. I saw a patient who had a pin with the point sticking out of the duodenal cap and buried in the head of the pancreas only the head remaining in the duodenum (Fig 52 b). After its removal a prolonged pancreatitis resulted. At first routine x-ray studies had failed to show this pin but later careful study showed it lying transversely in the epigastrium. X-ray and gastroscopic studies will often reveal the foreign body which in some cases may be removable through a long esophagoscope but in most cases will require surgical removal. In uncomplicated cases some recommend a period of watchful waiting to see whether the object will leave the stomach but the danger of its causing more trouble further down makes this a risky procedure.

#### TRAUMA

Injuries to the stomach as a rule belong entirely in the realm of surgery. The ones due to penetrating wounds such as bullet or stab wounds and to foreign bodies such as glass and metal objects swallowed in severe accidents are usually obvious. Supportive treatment for shock and hemorrhage and prompt operation are indicated. Compression injuries to the abdominal wall without penetration may cause tears in the stomach especially of the posterior wall. These occur almost always when the stomach is filled since the flexible elastic empty stomach is not easily injured in this way. The symptoms and signs of an acute perforation are usually present although in some cases the gastric wound may be spontaneously walled off and may later rupture secondarily. The usual procedures used for the determination of a ruptured viscus are indicated as is surgical care.

#### SPONTANEOUS RUPTURE

Spontaneous rupture of the stomach without injury has been described but is now rarely seen. It has usually occurred when the stomach was already distended and ruptured as a result of sudden liberation of gas by fermentation or by the carbon dioxide produced by sodium bicarbonate. Sudden increased intra-abdominal pressure as from labor has also been implicated. The signs and treatment are the same as for traumatic rupture.



Figure 52 *a* Bobby pin in stomach (scout film) *b* Pin in duodenum found during cholecystography showing (1) gallbladder dye filled (2) pin in duodenum found at operation to have penetrated into pancreas

spoons keys and other objects which the patients were hiding by swallowing them (5) Hair may be swallowed not only by mental patients but also as a result of habit in persons with long hair. The hair becomes matted and may form a large foreign body known as a trichobezoar or hair ball. Other bezoars may be formed from fibers of fruits or vegetables and are called phytobezoars, the most frequent example being caused by the sticky gum of persimmons (Fig 51 *a*). Others may be composed of mixtures of hair fibers, bismuth large pills or capsules, varnish and shellac (from drinking of alcoholic furniture polish).

*Large objects* such as bezoars and table silver may remain in the stomach for long periods causing few if any symptoms except a sense of fullness. Mental patients may be proud to demonstrate the clanking of silver by pounding the abdomen or by jumping up and down. Complications caused by the foreign bodies will cause symptoms. Gastritis and ulcer, stenosis and perforation will produce characteristic symptoms. X-ray films will disclose the cause. Operation is always required.

*Smaller objects* may remain in the stomach for longer or shorter periods depending upon the size or the degree of relaxation of the pylorus. I have seen a moderate cicatricial pyloric stenosis made intermittently complete by a collection of olive pits which could not pass through the pylorus and acted as ball valves, meanwhile causing a considerable degree of gastritis (Fig 51 *b*). Some might cause ulcerations or erosions and even perforations if they have sharp edges or protrusions. Blunt pins may cause no damage (Fig 52 *a*). The objects passing through

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# The Intestines

## General Discussion

Although the large and small intestines have different anatomical characteristics and different functions they form a part of the gastrointestinal tube whose general function of digestion and absorption of ingested food substances and excretion of unused remnants they carry on together. The fact that the same or similar diseases occur in both often simultaneously and produce similar symptoms makes it desirable to study the entire intestinal tract at the same time thus avoiding many duplications. The anatomy and physiology of each part will be discussed followed by a general discussion of symptoms affecting each and the diagnostic and therapeutic measures applicable to both.

## Anatomy and Physiology of the Small and Large Intestines

### THE SMALL INTESTINE

The small intestine is without doubt the most important part of the alimentary canal. Man can live without his esophagus, stomach and colon but not without his small intestine. Removal of one third or more of the small intestine is incompatible with life and removal of even less than this with a comfortable existence.

### Anatomy

There are certain anatomical facts about the small intestine which are important to remember. Its length varies from 20 to 25 feet. Its lumen becomes progressively smaller from duodenum to terminal ileum as does its blood supply and the thickness of its walls & change in accordance with the character of its changing contents. It is usually divided anatomically into three parts as follows:

The *duodenum* is the first 8 to 12 inches, the length varying according to the position of its four parts. The *first portion* also called the "cap" or bulb has already been described as being anatomically and physiologically more a part of the stomach than of the remainder of the duodenum. It is about 2 or 3 inches long and while usually pointing upward it may point straight out toward the left or even downward depending on the position or the degree of distortion of the stomach. It usually lies just below the neck of the gallbladder and when it is filled with food may on a plain x-ray film be mistaken for the gallbladder. Normally it is located behind the liver and may touch the transverse colon. It may be displaced by the head of the pancreas or by enlargement of retro

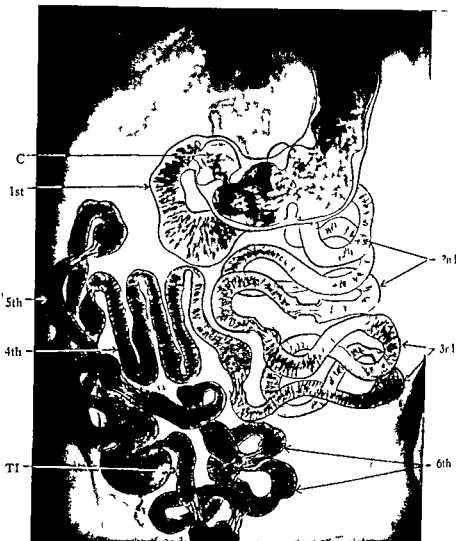
peritoneal lymph nodes. Occlusion of the end artery circulation of its anterior and posterior walls favors the development of areas of necrosis and ulceration the so called peptic ulcer. The *second or descending portion* makes a slight curve around the head of the pancreas to which it is attached. It lies close to the right kidney and ureter and the psoas muscle. Diverticula are frequent on its mesial aspect. The *third or transverse portion* running across below the stomach may be pushed downward by an enlarged head of the pancreas causing an enlargement of the normal loop formed by the second and third portions. The *fourth portion* runs upward to behind and sometimes above the mid portion of the stomach where if filled with barium it may hide or simulate a defect such as is caused by ulcer or cancer. It ends abruptly at its junction with the jejunum with which it forms an angle of varying degree. In a thin ptotic person standing may cause a marked accentuation of this angle producing more or less stasis.

The jejunum and ileum are not clearly defined. Their combined length varies from 20 to 25 feet approximately one third of which is jejunum two thirds ileum. Together they are called the mesenteric small intestine because they are covered by the fan shaped mesentery whose root lying posteriorly is only 6 inches long. The mesentery contains much fat protecting the rich blood nerve and lymphatic supply to the intestine. With the abdomen open the squirming active small intestine resembles a heterogeneous bunch of worms but when filled with barium and viewed under x ray its arrangement is fairly constant (Fig. 53). The jejunum usually lies in the left upper quadrant and groups of ileal coils can be seen in the right upper quadrant the right lower quadrant and finally more or less in the pelvis the terminal ileum crossing to the right to enter the cecum at its side. The ileocecal junction takes place practically at right angles and there is identifiable at this point a sphincter muscle as well as a valve the ileocecal sphincter and valve respectively. The terminal ileum has the smallest lumen of the small intestine and in conjunction with the valve and sphincter thus becomes a region in which foreign bodies may lodge or because of irritation fibrotic and neoplastic changes may take place.

The walls of the intestine consist of the usual layers. The jejunum and ileum are peculiar in that the mucosa and submucosa are thrown into plications the Kerkring folds and the mucosa consists of a tremendous number of elevations the villi an arrangement whereby the total mucosal surface is increased for the purposes of absorption. These configurations produce the characteristic feathery or herring bone appearance on x ray films.

### Physiology

The primary functions of the small intestine are digestion and absorption. For these to be accomplished it is necessary for the partially



**Figure 53** Small intestine Six groups of coils 1st duodenum 2nd upper jejunum 3rd lower jejunum 4th upper ileum 5th mid ileum 6th lower ileum C duodenal cap TI terminal ileum (Drawing made from x ray studies Lewis G Cole Radiographic Exploration of the Gastrointestinal Tract Bruce Publishing Co )

digested food entering from the stomach to be thoroughly mixed with digestive enzymes and transported through the intestinal lumen for absorption by the mucosa. Alvarez's gradient theory explains why material present at the upper end of the intestinal tract will automatically be carried to the lower end, entirely independent of nerve supply and therefore of nervous influences. To be sure, the autonomic and sympathetic nervous systems usually act as guides and controls of this process and may, if not properly balanced, cause disturbances in motility. The intestines rapidly get accustomed to such abnormal nervous influences,

however and are soon not affected by them. It is therefore rarely if ever necessary or desirable to cut off their vagus and sympathetic supply in order to overcome such disturbances.

The *speed of transit* from stomach to ileocecal valve is remarkable. Ingested material observed by x-ray examination usually reaches the ileocecal valve in one to three hours but is delayed there for purposes of absorption. Although the cecum begins to fill in three hours the small intestine is normally not entirely empty until eight hours after ingestion of a meal. According to Cole peristalsis is carried on by the muscularis mucosae; the muscular layer of the intestine is concerned with the rhythmic segmental contractions which mix the intestinal enzymes thoroughly with the food. Movements of the villi a wavy action bring the digested material into contact with the absorption surface. The mechanism at the ileocecal junction valve and sphincter regulates the passage of the contents according to the degree of absorption of liquid nutrient which has taken place while preventing reflux from the cecum. It also opens when more food enters the stomach. When there is increased pressure beyond as with obstruction in the colon or with an enema given under sufficient pressure cecal contents may regurgitate into the ileum. Thus regurgitation may extend upward to a distance of several feet permitting study of the barium filled ileum on x-ray examination. In extreme cases regurgitated material may reach the upper small intestine and the stomach and in exceptional cases may even be recognized in vomitus.

*Digestion* Although normally ingested foods have been somewhat prepared for small intestinal digestion by salivary and gastric enzymatic action and by heating, mastication and dilution the small intestine must carry digestion to the point at which the foods can be absorbed and used by the organism for growth, repair and maintenance. The external secretions of the pancreas and liver take part in the process aided and supplemented by the succus entericus which is produced by the intestinal glands of Lieberkuhn and Brunner. First the acid gastric contents must be alkalinized then the various enzymes take over. When pancreatic juice is cut off for a time the succus entericus can substitute for it to some extent.

*Carbohydrates* must be changed to the absorbable monosaccharides—dextrose, fructose, levulose or galactose. This is accomplished by the action of amylase or amylolysin, the enzyme which converts starches both cooked and uncooked to monosaccharides. More complex sugars such as sucrose (table sugar) and lactose (milk sugar) are changed to monosaccharides by invertase and lactase respectively. Upon reaching the liver the simple sugars are converted into glycogen and stored for future use in the liver and muscles.

*Proteins and peptones* are split by protease (trypsin) and other supple





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may be conical the appendix coming off at its lower end the lumen gradually tapering down to the tip of the appendix This is the fetal type It may be round and fairly small with unequal sacculations the infantile type or large with equal sacculations the adult type The ileum may enter it at different angles The ileocecal valve and sphincter are located here The appendix may come off at various points even posteriorly and may extend upward behind the cecum and ascending colon even to the subhepatic region and downward deep into the pelvis At the junction of the cecum and ascending colon a cecocolic sphincter has been postulated from x ray observation but has never been demonstrated anatomically

The *ascending colon* ends at the *hepatic flexure* which is practically fixed being only moderately movable with respiration The *transverse colon* may cross the upper abdomen in a straight line to the *splenic flexure* which lies under the diaphragm and is considerably higher than the hepatic flexure or it may dip downward to varying degrees in "U" or "V" form extending even into the pelvis in narrow asthenic persons An ill defined sphincter Cannon's ring is located at the junction of the first and middle thirds The fat gastocolic omentum hangs down in front as a protection from injury The *descending colon* extends 10 or 11 inches to the pelvic brim It is relatively fixed The *sigmoid* with its long mesentery makes a couple of turns and enters the rectum The sigmoid varies in length from a fairly small "S" to a long piece of colon resembling the descending colon and extending up as high as the splenic flexure It is easily pushed up with an enema and shrinks down after defecation The recto sigmoid junction is angulated and narrowed delaying the passage of feces This makes it a point of irritation for the development of neoplasm and ulcerations

The *rectum* is curved lying in the hollow of the sacrum and coccyx and then making two or three curves each guarded by transverse and oblique folds the valves of Houston from two to six in number The whole arrangement tends to control expulsion of feces The lower part of the rectum lies below the peritoneal reflexion and has no mesentery The upper part is covered anteriorly by peritoneum a pouch of peritoneum lying between the bladder or the uterus called the rectovesical pouch in the male the pouch of Douglas in the female reaching down anteriorly to within reach of a finger introduced into the anus A cancer implanted in it (Krukenberg tumor) can easily be felt The anal canal the last  $1\frac{1}{4}$  inches of the rectum is lined with squamous epithelium the rest of the colon with columnar epithelium A finger in the rectum can palpate the entire prostate anteriorly in the male the cervix and often the fundus and adenexa in the female

The blood supply of the right side of the colon is from the superior mesenteric artery and vein the left from the inferior The veins drain into the portal system Lymphatics run along the arteries with nodes at

mental enzymes into peptides (amino acids) which after absorption are resynthesized by the body into its own proteins. At times proteins are absorbed unchanged.

*Fats* are split by lipase into glycerol (glycerin) and fatty acids which when mixed with bile salts are emulsified and absorbed. When bile is not present more fats are found in the stool than when only pancreatic juice is prevented from reaching the intestine.

Indigestible material such as seeds, hulls, peels, strings, cellulose and foreign bodies able to pass the ileocecal barrier are carried to the colon for bacterial action and expulsion.

*Absorption* The intestinal mucosa normally is capable of absorbing only simple diffusible nonspecific substances, the end products of digestion. It appears as if unchanged proteins can be absorbed at times as when distant allergic reactions to specific proteins occur too soon after ingestion to have permitted of their digestion. It has even been suggested that direct absorption may have taken place in the mouth, esophagus or stomach so soon has a reaction been observed. The rapidity of absorption of the normal end products of digestion varies considerably, being dependent on the rapidity with which they are carried through the intestine, the degree of segmentation of the intestinal wall and the competition between their ingredients. Dextrose, mainly absorbed in the upper small intestine, has been thought to compete with amino acids, each slowing down the absorption of the other to a slight extent. On the other hand, some investigators have found that they enhance each other's absorption. Fats are always absorbed quickly and help to regulate osmotic pressure. For the absorption of vitamins, especially A, D, E and K, and of calcium, bile salts are necessary, which accounts for the evidences of vitamin deficiency in biliary obstruction. Absence of vitamins of the B group may produce diarrheas.

### THE LARGE INTESTINE

#### Anatomy

The colon is usually from 6 to 8 feet long, although variations in its length occur when its flexures are long and redundant. As seen by x-ray the colon may appear greatly lengthened and the flexures redundant, but it may shrink down to half the size within a short time. This variation is also seen in the diameter of the colon. Its greatest average diameter of about 3 inches at the cecum decreases gradually until it is only about 2 inches or less at the distal sigmoid. It differs from the small intestine in its long longitudinal muscle, its sacculations due to haustra and its appendices epiploicæ. Instead of a short thick mesentery, it has the *meso* colon surrounding the colon and meeting posteriorly where it encloses the vessels and nerves supplying the colon.

For purposes of description the colon is divided into four or five portions and three flexures. The *cecum* is the most variable portion. Its shape

elimination and then to store it so that it can be eliminated in an orderly fashion. The proximal half of the colon to Cannon's ring is concerned with absorption of water, salts, dextrose and other dialyzable substances; the distal half with storage and expulsion. Millions of bacteria of the enterococcal variety brought down from the ileum and of the acid or alkali producing varieties normally inhabiting the colon break down cellulose from the food forming histamine, indole, skatole, phenol, cresol and other compounds. None of the usual colonic bacteria produce toxins formerly held to make constipation dangerous.

**Secretion.** The mucus glands of the colon secrete mucus for the purpose of protection and for lubrication. Irritation from any cause—chemical, mechanical, bacterial and even nervous—causes an increase in secretion as a protective measure and possibly for the purpose of detoxification. With continued irritation as from habitual use of cathartics, enemas or colon irrigations, from ingestion of foods to which a patient is allergic, from chronic infection and to some extent from repeated nervous insults, the mucus not only increases in quantity but also becomes thicker. It may become either gelatinous, sometimes forming casts of a gummy consistency, or stringy or membranous, coming out in strings or patches of mucosa. As mucus contains from 8 to 10 per cent of nitrogen, excessive secretion of mucus may cause considerable depletion of body protein.

**Absorption.** Aside from the materials already mentioned as being absorbed in the proximal colon, the remainder of the colon absorbs water sufficient to solidify the contents to the consistency of feces. It can also absorb some drugs instilled into the rectum such as opium and belladonna and their alkaloids, some antibiotics, alcohol, aniline dyes, calcium chloride and others, as well as dextrose and amino acids. Formerly much was written about "intestinal toxemia" thought to result from constipation, but no proof of toxic absorption through a normal mucosa was ever established. I have known patients to go from two to four weeks without defecation and experience no toxic symptoms. Many healthy persons enjoy one bowel movement a week giving Sunday morning for the enjoyment of leisurely defecation.

**Motility.** The transport of contents through the colon is not a direct continuous passage from cecum to rectum. It is guided by the physiologic requirements. The liquid material entering through the ileocecal orifice gradually fills the colon to Cannon's ring, whose sphincter action keeps it in the proximal colon to permit absorption. This part of the gastrointestinal tube is also unique in that normally reverse peristalsis takes place, the content being moved up and down to facilitate absorption. When sufficient water has been absorbed or when a wave of small bowel peristalsis following ingestion of food reaches the cecum, peristaltic "rushes" begin at the cecum and push the contents rapidly across the transverse colon. At times the contents are allowed at first to rest at the splenic

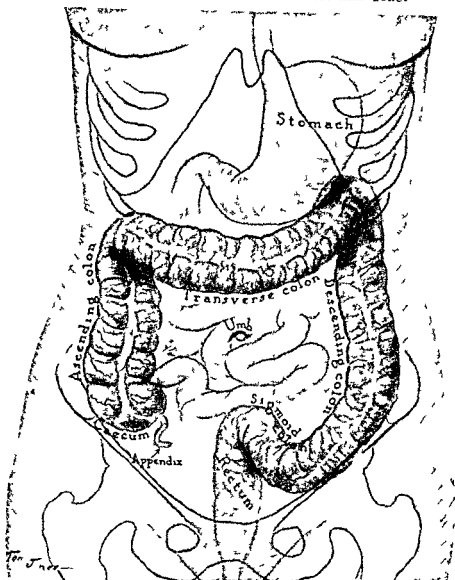


Figure 54 Colon 1 Location of Cannon's ring 2 ileocecal sphincter (Courtesy of Wyeth Laboratories)

intervals especially in the ileocecal region and under the flexures. The nerve supply is still under dispute. Some contend that the colon receives fibers from the vagus nerves; others deny it. Sympathetic stimuli are inhibitory; the vagal would be stimulating.

### Physiology

The colon receives from the ileum the material which the stomach and small intestine have not digested or absorbed. Its function is to make use of any parts of these materials it can to render the waste material fit for

## Pathology of the Large and Small Intestines

The intestines may show various anomalies which are usually associated with anomalies elsewhere such as cleft palate club feet congenital hip dislocation transposition of viscera and others Diverticulosis occurs in both the large and small intestines with some difference in complications Poisonings and inflammations usually affect both as do many acute infections Some infections affect either or both the pathological findings differing because of differences in structure Allergic reactions may occur in various parts or in the whole intestinal tract Parasites may infest parts of or the whole intestinal tract Neoplasms both benign and malignant may occur in any portion of the intestines either as lone tumors or as multiple tumors involving different parts of the intestine at the same time It is therefore imperative to study the entire length of the intestines when a tumor is found at one point The intestines are also affected in differing degree by general infections and by cardiovascular renal hepatic and neurological conditions Trauma both internal and external may affect different parts of the intestines in varying ways A recent cause of intestinal disease is the increasing use of the broad spectrum antibiotics These even in small doses taken for but a short time may cause irritation varying from a slight reddening and edema to a severe pseudomembranous or hemorrhagic enterocolitis The cause may be an allergic reaction to the drug or to the growth of resistant organisms such as staphylococci or molds

## Symptomatology and General Treatment of Symptoms

Certain general symptoms can be grouped together for better understanding They are due primarily to the effect of disease on intestinal physiology They include pain of varying kinds and degrees distention retrostaltic symptoms vomiting diarrhea and constipation Each will be considered in turn and its treatment briefly discussed

### PAIN

Pain is the most obvious symptom The term includes varying degrees of pain from mild distress to severe agonizing pain or tenesmus The location of the pain its mode of onset and its type must be considered It must be realized that in many instances a patient may with great accuracy describe each of these factors and frequently can also indicate the nature of a lesion by describing a feeling of narrowing at a certain point distention behind it and the passage past it of gas or intestinal content as a result of relaxation A careful personal history not just hearsay from attendants is often very rewarding

### Location

Although in general midabdominal pain is associated with small intestinal lesions and suprapubic or lateral pain with those of the colon

*flexure* to be pushed farther with subsequent rushes or they may be pushed directly to the sigmoid there to be stored until ready to be expelled into the rectum and out of the anus usually on the following day. These mass movements are accomplished by a combination of successive tonus changes, haustral contractions and movements of the muscularis mucosae. They are most active in the day, infrequent and milder during the night. Sudden emotion may accelerate or inhibit them.

*Timetable* Usually it takes twenty-four hours for the residue from a meal to reach the sigmoid, then in the form of a fairly firm mush. Water continues to be absorbed and at the end of another eighteen to twenty-four hours or longer if the contents are small in amount because of insufficient ingestion of indigestible material the contents are firm and ready to be expelled. An increase in activity such as arising and exercising after a night's rest starts peristaltic waves throughout the gastrointestinal tube. A further stimulus is obtained from eating or drinking. The waves then become strong enough to push the sigmoid contents into the rectum distending it. The rectum is insensitive to tactile or chemical stimulation but distention can be felt and causes a desire to defecate. The involuntary peristalsis is aided by voluntary contractions of abdominal muscles, the diaphragm and the levator ani and is further aided by bending of the body forward. There follows a relaxation of the sphincter ani causing the mass coming down from the sigmoid to be expelled as formed feces. The desire to defecate caused by distention of the rectum at first is mild and may be controlled and lost but recurs with each further arrival of feces. It may be so insistent that it finally cannot be controlled or it may be controlled voluntarily. Feces allowed to remain in the rectum becoming increasingly dehydrated may at times reach the point of fecal impaction. To maintain normal function it is desirable to heed the call of nature promptly although trying to establish a habit of defecation at a certain convenient hour each day preferably after breakfast can be induced by stimulating defecation for a few times by the insertion into the rectum of a dilator or a simple suppository of cocoa butter. Anything introduced into the rectum produces the defecation reflex.

*Summary* It can now be realized how well adapted the gastrointestinal tract is to its requirements. Ingested materials are carried from the mouth independent of voluntary control and guided only by involuntary nervous control. Delays occur at the cardia to permit food to enter the stomach slowly, at the pylorus to permit proper preparation of food for intestinal digestion, at the ileocecal valve to prolong absorption of the food digested in the small intestine, at Cannon's ring to promote further absorption of liquids, at the sigmoid to allow for storage and formation of the waste material in the rectum to prevent hasty expulsion and finally under voluntary control to avoid embarrassment.

this type of distention. Aside from air other gases swallowed or some produced in the gastrointestinal tract by chemical action or fermentation may distend the bowel causing a feeling of fullness moderate pain flatulence or "blowing up." Such gases may be entirely absorbed through the intestinal mucosa or may not be absorbed producing increasing distention. Normally they are finally expelled as flatus. Persistent distention of the small or large bowel is suggestive of an "acute abdomen" described on page 59. If the distention is midabdominal and presents a ladder like arrangement indicating a distended small intestine an obstruction of the ileum or proximal colon must be suspected. Distention of the right abdomen or ascending colon suggests obstruction of the pelvic colon. Acute generalized distention is seen in intestinal obstruction and in peritonitis from any cause. Chronic persistent colonic distention and elongation is often called acquired or secondary megacolon and is accompanied by thickening of the colonic wall (p. 333). It is usually secondary to organic obstruction of the distal colon although occasionally ascribed to the habit of neglecting normal bowel function. Gases may often be successfully removed by suction through a Miller Abbott tube but will rapidly recur if real obstruction is present.

Intestinal distention has been postulated to occur as a purely functional disorder due to paralysis of intestinal musculature and has been called "paralytic ileus." The term is unfortunate in that it encourages the making of the easy diagnosis when careful study might reveal an organic cause. *True paralytic ileus* occurring in paraplegic patients during any acute pain or shock or as a result of inflammation in connection with gallbladder or genitourinary disease after laparotomy or even in pregnancy is classed as functional. There is secondary relaxation of the intestinal musculature with blocking of circulation.

Observation of the arrangement or location of gas accumulation may be a great help in diagnosis. Observation or palpation and percussion of midabdominal distention suggest lower small intestinal obstruction. Right sided distention often occurs with sigmoid or rectal obstruction.

### X-ray Examination

X-rays are a greater help. Dilatation of the duodenum indicates obstruction of the duodenum or jejunum usually at the duodenojejunal angle. Small intestinal dilatation even when distended to the diameter of the colon can be recognized by the transverse striations of the valvulae conniventes the Kerkring folds and the transverse arrangement of the intestinal coils producing the ladder like appearance. It is significant of obstruction in the lower small intestine or cecum. Distention of the proximal colon to Cannon's ring occurs as a result of spasm at that sphincter due to obstruction of the pelvic colon. The whole or greater part of the colon becomes distended later when the sphincter fails. Colonic distention due to obstruction elsewhere stops abruptly at the



variations in location are so frequent that it is dangerous to draw conclusions from location alone. The hunger pains of ulcer usually epigastric may be manifested over another lesion such as sigmoid diverticulitis or appendicitis. Pains from pelvic urological biliary or even cardiac diseases may occur in the same regions as intestinal pain.

### Onset

The mode of onset may be of importance. Sudden onset occurs in an acute abdominal calamity perforation or obstruction in food or other poisoning in dysentery and trauma. Gradual onset occurs in milder or more chronic intestinal diseases.

### Character

The character of pain is of greatest significance. The exact mechanism of pain production is not clearly understood. It is known that the intestines down to the anal canal are insensitive to pain. Under local anesthesia of the abdominal wall and peritoneum the intestines can be handled incised resected and anastomosed without causing pain except when unduly pulled upon or distended. Peristaltic pain is recognized as intermittent cramplike pain tenesmus or griping pain usually accompanied by diarrhea or at least a frequent desire to defecate. It also occurs with obstruction. Peritoneal pain is added when there is much distention with stretching of the intestinal wall. Sudden agonizing pain occurs with sudden stretching bursting or tearing of the peritoneal coat from internal or external ulcerations or trauma. When peritonitis supervenes the pain is severe and constant the abdominal muscles become rigid and there is a cessation of peristalsis the silent belly.

### Treatment

Pain should not be treated specifically until a diagnosis has been established or an operation decided upon. As a rule treatment of the cause of the pain should relieve it.

### ABDOMINAL DISTENTION

Abdominal distention the protrusion of the abdominal wall resembling distention with gas is often seen in neurotic patients. They make the abdominal wall protrude by pushing out the abdominal muscles usually also swallow air and belch and ask for relief from "gas". If they are told to stand erect open the mouth and draw in the abdominal wall the apparent distention can be made to disappear. Or with the patient lying supine a scaphoid abdomen may be demonstrated. It is possible however for a patient to swallow sufficient air (p. 9) to cause marked gastric distention so that the large air filled stomach can be seen and then the air when not belched to go through the pylorus causing ileus the distention of small and large intestines with air later expelled as flatus. Prevention of air swallowing will soon cause a disappearance of

this type of distention. Aside from air other gases swallowed or some produced in the gastrointestinal tract by chemical action or fermentation may distend the bowel causing a feeling of fullness, moderate pain, flatulence or "blowing up." Such gases may be entirely absorbed through the intestinal mucosa or may not be absorbed producing increasing distention. Normally they are finally expelled as flatus. Persistent distention of the small or large bowel is suggestive of an "acute abdomen" described on page 59. If the distention is midabdominal and presents a ladder like arrangement indicating a distended small intestine, an obstruction of the ileum or proximal colon must be suspected. Distention of the right abdomen or ascending colon suggests obstruction of the pelvic colon. Acute generalized distention is seen in intestinal obstruction and in peritonitis from any cause. Chronic persistent colonic distention and elongation is often called acquired or secondary megacolon and is accompanied by thickening of the colonic wall (p. 333). It is usually secondary to organic obstruction of the distal colon although occasionally ascribed to the habit of neglecting normal bowel function. Cases may often be successfully removed by suction through a Miller Abbott tube but will rapidly recur if real obstruction is present.

Intestinal distention has been postulated to occur as a purely functional disorder due to paralysis of intestinal musculature and has been called "paralytic ileus." The term is unfortunate in that it encourages the making of the easy diagnosis when careful study might reveal an organic cause. *True paralytic ileus* occurring in paraplegic patients during any acute pain or shock or as a result of inflammation in connection with gallbladder or genitourinary disease after laparotomy or even in pregnancy is classed as functional. There is secondary relaxation of the intestinal musculature with blocking of circulation.

Observation of the arrangement or location of gas accumulation may be a great help in diagnosis. Observation or palpation and percussion of midabdominal distention suggest lower small intestinal obstruction. Right sided distention often occurs with sigmoid or rectal obstruction.

### X-ray Examination

X-rays are a greater help. Dilatation of the duodenum indicates obstruction of the duodenum or jejunum usually at the duodenojejunal angle. Small intestinal dilatation even when distended to the diameter of the colon can be recognized by the transverse striations of the valvulae conniventes, the Kerkring folds and the transverse arrangement of the intestinal coils producing the ladder like appearance. It is significant of obstruction in the lower small intestine or cecum. Distention of the proximal colon to Cannon's ring occurs as a result of spasm at that sphincter due to obstruction of the pelvic colon. The whole or greater part of the colon becomes distended later when the sphincter fails. Colonic distention due to obstruction elsewhere stops abruptly at the

ing this speedy progress may be produced by a great variety of conditions and diseases but the common factor in all is irritation. The mucosa or the neuromuscular mechanism may be irritated as follows

### INTESTINAL IRRITATION

#### Mucosal Irritation

The causes of mucosal irritation may be listed as follows

1 *Infection* Bacteria viruses or mycoses or their products either floating in the contents or attacking the mucosa of the intestine and of diverticula causing inflammation or ulceration—examples are enteritis colitis diverticulitis and specific infections such as bacillary dysentery food poisoning typhoid salmonellosis tuberculosis cholera lympho granuloma venereum actinomycosis blastomycosis coccidioidomycosis and other infections

2 *Parasites* by simple mechanical irritation by chemical irritation from their secretions or by mucosal invasion. Examples are worms flagellates amebae and others. The elimination of these parasites is discussed elsewhere (p 134)

3 *Undigested Foods* These are found as a result of (a) failure of gastric digestion as in achylia gastrica or after operations on the stomach (b) failure of digestion by pancreatic ferments in pancreatic disease (c) failure of fat digestion due to decrease or absence of bile (d) failure of digestion after operations on the gastrointestinal tract and appendages

4 *Unabsorbed foods* particularly fats as in sprue "malabsorption" syndrome celiac disease and Whipple's disease

5 *Specific foods* to which the patient may be allergic as in regional ileitis and ulcerative colitis. Foods which are inherently poisonous such as mushrooms and overeating of fruits may also be a cause

6 *Chemicals* either directly ingested as in accidental or suicidal poisoning by cleansing fluids bichloride iodine or match heads. Excretion into the stomach or intestine of such chemicals as mercury and other metals iodine or arsenic may also cause diarrhea

7 *Toxins* exogenous produced directly by bacteria viruses or mycoses or by their action on the tissues or endogenous as in thyrotoxicosis uremia acidosis and adrenal insufficiency

8 *Antibiotics* especially those of the broad spectrum group which act either as allergens or by destroying some bacteria and permitting others resistant to the antibiotics to develop and cause the diarrhea

9 *Laxatives* whose habitual use promoting loose stools really produces a diarrhea when patients are complaining of constipation. Some like cascara frequently also cause pigmentation of the mucosa

10 *Foreign bodies* such as glass pins toothpicks and other articles accidentally or intentionally ingested and passing the natural barriers above

## Neuromuscular Irritation

This will include some causes affecting also the mucosa the question then being which is more affected. The principal causes are

1 *Nervous system diseases* such as tabes dorsalis, paralysis and paraplegias which act upon the musculature particularly. General visomotor instability may produce diarrhea or constipation.

2 *Psychic disturbances* such as fear, anger and other emotional upsets which are generally recognized as causing acute diarrheas but whose effect in causing chronic diarrheas is questionable.

3 *Neoplasms* both benign and malignant. When small they act by irritating the mucosa producing the effect of a foreign body, when large by disorganizing the entire wall and causing either constipation or diarrhea or both alternately.

4 *Extraneous diseases* including female pelvic inflammation or tumors, prostatic inflammation or hypertrophy and diseases of the urinary tract from urethra to kidney may cause irritation by direct external contact or pressure or reflexly. By inducing pressure and spasm these conditions may also cause constipation.

5 *Passive congestion* of cardiac or portal origin which may also cause constipation as well as diarrhea.

6 *Motility disturbances* caused by peptic ulcer at times by chronic biliary tract diseases, by chronic appendicitis, by diverticulitis, by amyloidosis and by chronic nephritides must be included.

It will be seen from the foregoing merely partial lists that a large number of conditions can be responsible for diarrhea. The two lists contain sixteen general classifications and under each one there are mentioned from two to ten subclassifications or diseases. It is therefore safe to say that there are well over 100 possible causes of diarrhea. Upon being consulted by a patient with diarrhea it is important to avoid purely empirical symptomatic treatment. Such treatment may not only not help the patient but may actually do him incalculable harm by delaying adequate diagnosis and specific treatment.

## DIAGNOSIS

A careful history is important. In general diarrhea is usually though not always accompanied by cramps and tenesmus. Frequent liquid stools result in rapid dehydration and loss of weight and strength and the constant desire causes nervous depression. Frequent liquid irritating stools cause more or less anorectal discomfort, excoriation, edema and the development of pruritus, fissures and hemorrhoids. Some of the symptoms are more prominent than others but the variations are not characteristic of any particular cause except as to their relationship to symptoms of the primary cause.

### Acute Diarrheas

If there is a history of previous similar attacks at varying intervals allergy may be suspected. Some infectious diseases such as the dysenteries may also occur intermittently.

If the patient can remember no previous diarrhea except a few clearly due to food poisoning with other members of his family likewise affected the present acute diarrhea may be due to an acute infection or poisoning or it may be the beginning of a chronic diarrheal disease. Either may or may not show fever, general prostration or retrostaltic symptoms. Diarrheas of only a few days' duration may be due to a virus infection to staphylococcus toxin to a mild *Salmonella* infection to a newly developed allergy in a patient with other allergies to a poisonous food such as mushrooms or to an inorganic or organic poison taken intentionally or accidentally. A careful history may give a clue as to the cause although this may be deceptive especially since an acute food poisoning may be the starting point of a more serious infection. Acute appendicitis may start with a diarrhea and should be ruled out before making a diagnosis of food poisoning. In hot weather foods and sauces which have been standing a long time acting as culture media especially in restaurants or bakeries may be suspected of harboring pathogenic organisms. Any foods not actually hot enough to destroy bacteria may become contaminated by food handlers who are infected and are careless about washing hands after a diarrheal stool or who have an infected finger or a nasal discharge (staphylococcus toxins mixed with food). Careful questioning and examination of containers which show residues of poison may disclose such a poisoning. (See Food Poisoning page 71)

Intestinal obstruction high up or an intussusception at any point may be early associated with diarrhea or a bloody discharge (p. 67)

### Chronic Diarrhea

When diarrhea continues for more than a few days one of the other causes mentioned before may be the cause. Careful search by history, physical examination, laboratory and x-ray studies should be undertaken to make an exact diagnosis so that a rational treatment may be instituted.

#### PHYSICAL EXAMINATION

In general dehydration, emaciation, pallor and general weakness may be more or less marked. A careful examination from head to foot should be done in every case to discover any of the causes mentioned before or to rule them out. No system should be neglected, nothing taken for granted. Abdominal examination may show no abnormal findings, may disclose distention, tenderness, muscle guarding or actual rigidity and evidences of an acute calamity. The finding of a mass will be significant. Skin lesions may be characteristic. Careful rectal examination and proctoscopy with or without biopsy may show only the effects of the diarrhea.

or may show the cause such as neoplasm lymphogranuloma bacillary dysentery amebic ulcers or ulcerative colitis. Neurological examination is necessary. Time spent on a complete physical examination may obviate many mistakes and permit of early specific treatment.

### SPECIAL DIAGNOSTIC PROCEDURES

Not all the following tests may be necessary but in some cases all may be required before a diagnosis can be made.

#### Laboratory Examinations

**Blood** Leukocytosis and an increased sedimentation rate in acute infections eosinophilia in parasitosis and allergy the characteristic findings in pernicious anemia hyperazotemia in renal damage hyperglycemia in diabetes positive cultures in septicemia are but a few significant findings in the blood. Agglutination tests are an aid in some diseases.

**Urine Examination** Pyuria hematuria glycosuria bilirubinuria and urobilinogenuria are important findings in diarrheas.

**Function Tests** Renal hepatic pancreatic cardiac thyroid and adrenal function tests may be a help.

**Fractional Gastric Analysis** Gastric analysis after histamine injection may disclose achylia.

**Skin Tests** For allergy tuberculosis lymphogranuloma salmonella mycoses and other organisms skin tests are often indicated.

**Stool Examinations** Repeated examination of stools is imperative in any case acute or chronic and is discussed in a general way on page 223. The physical characteristics of the stool may be significant. Liquid stools are usually considered necessary for the diagnosis of diarrhea but soft mushy stools or at times even slightly formed but frequent stools may occur. Some stools which are characteristic in appearance include the pea soup stools of typhoid the rice water stools of cholera the stools with blood and pus in bacillary dysentery the bloody mucoid stools of amebic dysentery and the frequent bloody discharges often without feces of ulcerative colitis and cancer.

**COLOR OF THE STOOLS** The color varies in different conditions. One can distinguish the normal dark brown in early acute diarrheas the green of rapid peristalsis from the upper small intestine down due to unchanged bile the red color of fresh blood the black stool of tarry consistency due to blood from the mouth to the duodenum the light yellowish color of small intestinal hypermotility the light gray clay colored stool of jaundice and colors of various kinds from ingested foods or medications such as black from iron and some berries red from beets or tomatoes green from green vegetables and orange from insufficiently chewed carrots.

**ADMIXTURES** *Blood* if present may be black from bleeding high up in the small intestine red and mixed with feces from the ileum and

proximal colon relatively little mixed from further down bright red and smeared on the outside of a stool or in clots from the rectum and sigmoid or bright red and seen only on toilet paper from the anal canal

*Mucus* appears in all diarrheal stools. It may appear as a coating on more or less formed feces as small jelly like masses mixed with feces or in the form of strings or pieces of membrane

*Pus* may be mixed with feces and hard to differentiate from thin feces except under the microscope or may be mixed with blood and mucus

Excessive amounts of *fat* will appear as a result of two principal causes failure of digestion and failure of absorption usually a combination of both. In failure of digestion by pancreatic juice or insufficient quantities of bile reaching the intestine as in obstructive jaundice neutral fats can be recognized by the Sudan III stain which stains the globules a deep red. The stools are bulky and buttery or oily and on standing may show unhydrolyzed fat on their surface like a gravy. The stools of sprue or malabsorption are homogeneous and greasy and owing to no failure of fat digestion show mostly fatty acids and soaps on microscopic examination

*Foreign bodies* may have to be screened out by passing the feces usually diluted with water through a sieve

*Undigested food* may at times be recognized grossly when there is a rapid progress through the bowel. Too much stress however is often placed upon the finding of shells, hulls, skins, seeds and leafy vegetables remnants of which are not normally digested and are usually found in any normal stool. The finding of such specific food remnants can however be a guide to the rapidity of progress when they have been known to be ingested once and only at a given meal

Worms may be discovered by the patient in stools or may be seen separately. Study for *parasites* and ova is described under Intestinal Parasites (p 134)

**ODOR OF THE STOOLS** The odor is not usually characteristic although some odors are suggestive. The foul odor in steatorrhea, the putrid odor from cancer, the sour odor of the gassy stools of carbohydrate fermentation and the pungent odor in amebic dysentery are fairly constantly detectable

**MICROSCOPIC EXAMINATION** A search for *parasites* and ova may be rewarding. As mentioned in the chapter on parasites microscopic examination may be necessary to distinguish the heads of the larger worms to differentiate ova and in the case of microscopic parasites to establish their nature. Preparations should be made for *cytological* study since cancer may be demonstrable. Pus and red blood cells and mucous shreds can be seen. Starch granules are recognized by their staining blue with iodine meat fibers by their transverse striations fat globules are observed as translucent globules which stain deep red with Sudan III fatty acid crystals as sheafs of fine crystals

In suspected poisoning stools as well as vomitus suspected foods and containers should be submitted to a public laboratory for toxicological and cultural studies

### X-ray Examination

In acute cases scout films may give a clue as to whether an acute abdomen is present due to perforation of a hollow viscus or an obstruction from any cause (See Acute Conditions of the Abdomen page 59)

In any chronic case a complete gastrointestinal study should be made Barium enema study should be done first to rule out neoplasms of the colon intussusception or other obstruction diverticulitis or colitis If there is no obstruction a detailed gastrointestinal series after an opaque meal with cholecystography is necessary to rule out the various lesions which have been mentioned as possible causes of diarrhea and incidentally to check on the rapidity of transport of barium Chest urological or other x-ray studies may be indicated

### TREATMENT

#### Prophylaxis

This consists in attention to sanitation to prevention of pollution of water and foods to isolation of infected persons and carriers and to preventive inoculation of persons going to endemic areas Inspection of foods restaurants and food handlers is important Equally so is adequate care of the many other conditions which may cause diarrhea

#### Symptomatic Treatment

Empirical symptomatic treatment should be avoided if possible since it may disguise the symptoms of a serious condition such as acute appendicitis diverticulitis or cancer A smooth diet such as an ulcer diet can do no harm unless the diarrhea is due to allergy to milk or some other constituent of that diet In acute diarrhea if an "acute abdomen" can be definitely ruled out 1/2 ounce of castor oil by eliminating the cause of irritation will often be a help Kolin and pectin separately or together may be taken freely to soothe the mucosa Oil or gelatin enemas may help Antispasmodics are of little if any value Opiates should be avoided unless tenesmus is severe and then used sparingly Paregoric 1 teaspoonful every three or four hours is usually helpful This subject is discussed under Acute Gastroenteritis and Food Poisoning (p 71) In chronic diarrhea the same treatment may help temporarily but will be of no permanent value The most important consideration is the diet which should be well balanced and nutritious should contain no food to which the patient is allergic and should be fortified with vitamins and minerals The diet must be modified according to the indications for the disease causing the diarrhea Hygienic care rest sunshine and fresh air must not be neglected



## Specific Treatment

When *parasites* have been identified suitable parasitocides are indicated

In *bacterial infections* sulfonamides or antibiotics tested beforehand to determine their effect upon the specific bacteria should be used Today there is a tendency to use synergistic combinations of antibiotics Antibiotics should be used cautiously because they themselves may cause diarrheas In some cases bacteriophages may be of great value

When broad spectrum *antibiotics* have been shown to be the cause of the diarrhea they should be stopped Newly tested ones may be substituted but must also be watched carefully When mycotic infection such as moniliasis can be demonstrated mycostatin is indicated This type of diarrhea is often helped by a diet containing buttermilk or koumiss If antibiotics are absolutely indicated a combination of mycostatin with a new antibiotic should be tried

In *virus infections* the treatment must be largely symptomatic since no specific agent to destroy viruses is known Secondary bacterial infection will require treatment as above

*Causative treatment* is most important Specific care of any or all of the diseases mentioned as being causes of diarrhea should be thoroughly carried out Some require only medical treatment others surgical These forms of treatment are discussed elsewhere

Psychiatric care may be indicated

## SUMMARY

It must be emphasized that diarrhea is a symptom It may be caused by many diseases some very serious It should therefore not be treated empirically but a careful study to determine its cause or causes should be instituted so that specific treatment can be carried out As in any other diseased condition it is important that adequate nutrition be maintained by balanced diet vitamins and minerals

## CONSTIPATION

Much has been written about constipation Its causes have been defined many symptoms and diseases have been ascribed to it many different kinds of treatments have been prescribed and medical and lay journals and newspapers are filled with advertisements of cures Yet no one has formulated a satisfactory definition of constipation Bowel movements insufficient in frequency or volume and fecal dehydration are the characteristics usually considered but without any standard established Accompanying symptoms of rectal fullness or actual irritation from the presence or expulsion of the hard feces and hemorrhoids from pressure on *lower hemorrhoidal veins* are usually described Retrostaltic symptoms toxic symptoms and neurological symptoms such

is pains in various parts of the body, headaches and tachycardia have also been attributed to this uncertain symptom.

A study of the physiology of the colon has demonstrated its condenser action, its carrying of contents by means of peristaltic rushes to the sigmoid and its storage there of the feces until they have attained sufficient bulk to be pushed into the rectum.

It has been shown by x ray studies that the unused material of ingested food reaches the sigmoid in twenty four hours and is usually not expelled for at least forty eight hours. The progress of a barium meal can be followed by x ray, but unless a standard diet is given during the x ray study, no very definite conclusion can be reached about abnormal delay of its passage through the gastrointestinal tract. We have always used a standard diet later described as an "anticonstipation diet." It is surprising to see how often "constipated" patients show normal progress when on this diet. The finding of an easily identifiable substance such as charcoal, aniline dye or small beads has been used to determine speed of evacuation.

### Questions about Constipation

When a person empties his colon by means of laxatives or enemas every day or even every other day, so that no normal forty eight hour accumulation is permitted, is it fair to say he is suffering from "constipation"?

The size of the fecal mass is of course dependent upon the amount of indigestible waste material, usually cellulose, that has been eaten. When a person eats little or no cellulose containing foods such as leafy vegetables and fruits or berries and their skins and seeds, why is it "constipation" if he does not have a bowel movement for several days or a week or until he has accumulated a sufficient bulk of feces?

If the consistency of the fecal mass depends upon the amount of liquids taken in excess of what is required for urine perspiration and water vapor in the breath, is it "constipation" when a person drinks little or no water and the stools are dry and hard?

When the normal lubricant mucus has been repeatedly washed out with cathartics or irrigations and consequently very little mucus or a sticky or stringy mucus has resulted, why should the resulting failure of easy defecation be labeled "constipation" or the abnormal mucus be attributed to mucous colitis?

We know that exercise, especially of the abdominal wall, helps to maintain normal tone of the colonic muscles, as well as the voluntary muscles helping in defecation. If a person has spent some time in bed or otherwise has taken insufficient exercise and has weak muscles, why speak of "constipation"?

The fecal mass is pushed into the rectum from the sigmoid as a result

of waves of peristalsis usually originating with the ingestion of a meal especially in the morning. If a person habitually eats no breakfast or very little and the stimulus is insufficient is this "constipation"?

The stimulus to empty the fecal mass from the rectum a combined involuntary and voluntary act, is produced by the sensation of a mass in the rectum. When the desire to defecate is suppressed repeatedly so that the sensation is lost with resultant tendency to the accumulation of feces making defecation difficult (dyschesia) why call this "constipation"?

When certain foods to which a person is allergic cause enough colonic and especially rectal spasm to delay the passage of feces should this be called allergy or constipation?

Most or all of the factors mentioned above are frequently neglected by a nervous busy careless person who then speaks of suffering from constipation. Is this fair?

The failure of normal defecation from neglect of any or all of the factors described with no evidence of an organic cause has been called functional or *habitual constipation* to distinguish it from the type due to organic narrowing from any cause called *organic constipation*. When so called constipation is due to anorexia nausea vomiting and insufficient food intake resulting from congenital megacolon inflammation edema infiltration cicatricial contraction kinking or intrinsic or extrinsic tumors is it rational to call it habitual constipation either for descriptive or therapeutic reasons?

So called constipation is a symptom. It should not be treated as a disease. The so called toxic symptoms attributed to constipation have been shown to be due either entirely to imagination or to mechanical pressure of a mass in the rectum and not to absorption of any toxic substances. This is easily demonstrated by placing a lamb's wool tampon in the rectum and when toxic symptoms occur pulling it out by its string instantly relieving the symptoms. There are no symptoms connected with simple constipation except possibly a full feeling. Its cause should be determined since it may be important especially if it is of recent occurrence. Extreme colonic dilation sometimes called acquired megacolon may result from long neglect.

### CHRONIC CONSTIPATION

In chronic cases a detailed history may disclose one or more of the errors of omission or commission mentioned above and their correction can be expected to result in permanent cure though this may take some time. It is best even then to make a complete gastrointestinal study to have the pelvis and prostate checked and the patient's general condition evaluated before starting any treatment.

When an organic cause has been ruled out I explain to the patient what is causing his constipation and what he must do to overcome it.

I do not like to temporize by allowing any laxatives although in an elderly patient a little mineral oil by mouth ( $\frac{1}{4}$  to 1 tablespoonful at bedtime) may be used for a short time. The recently introduced wetting agent "dioctyl sodium sulfosuccinate" manufactured under various trade names is harmless and of some value in keeping stools moist. It is not a laxative and its effect is not always permanent. It must not be used as a substitute for the diet and other measures recommended. A small impaction may be removed and normal rectal tone restored by a small retention oil enema the patient lying on the left side while 4 to 6 ounces of vegetable oil are instilled into the rectum at bed time and left in all night or even a few ounces of water may accomplish the purpose. Sometimes dilatation of the anal sphincter by a finger or dilator will be of value. Increasing exercise especially of the abdominal muscles must be taken. For a time until a normal bowel habit has been established a well balanced diet with a slight excess of cellulose residue wrongly called "roughage" is of value. I prefer the term "smoothage" or "bulk" for after the cellulose has been chewed and has passed through the gastrointestinal tract it is practically never rough. Such a diet would be as follows:

#### Anticonstipation Diet

<i>On Arising</i>	Water 1 or 2 glasses
<i>Breakfast</i>	Whole grain cereal with milk and sugar Egg 1 or 2 soft boiled or poached Bread whole wheat and butter 1 pat Milk 1 glass Fruit raw or cooked with skins and pulp
<i>Mid morning</i>	Water or milk fruit juice or fruit and whole wheat crackers or bran muffin
<i>Lunch</i>	Egg cheese or fish Vegetables cooked 2 or more kinds especially green Vegetables raw lettuce raw cabbage celery tomatoes etc Bread whole wheat and butter Milk 1 glass Fruit stewed or raw
<i>At 1-afternoon</i>	Same as mid morning
<i>Supper</i>	Same as lunch varying the vegetables and allowing meat
<i>At bedtime</i>	Same as mid morning
<i>Water</i>	At least 5 or 6 glasses a day

Additional bulk in the form of agar bran or the hydrophilic colloids obtained from psyllium and other seeds may be of value but an excess can easily be taken and occasion some blockage as the result of over filling of the intestinal lumen.

Vitamins especially those of the B complex are definitely indicated. Vitamin and mineral capsules can be obtained which will take care of both necessary factors.

If the regimen just prescribed is faithfully carried out patients with habitual chronic "constipation" will usually begin within a week or ten

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to interfere with bowel movement. The indications for treatment are usually obvious.

4 Some *endocrine dyscrasias* especially hypothyroidism and hyperparathyroidism are often associated with severe constipation and require appropriate treatment.

5 *Actual obstructions* of various degrees from bands, kinks, cicatricial stricture, diverticulitis, anomalies such as congenital megacolon and from cancer must be discovered and cared for appropriately.

#### SUMMARY

Constipation is a symptom and should never be treated *per se*. Before embarking on any line of treatment a complete study should be carried out in order to make sure that no organic disease, especially cancer, is being overlooked and its prompt care neglected. In the cases not due to organic disease, errors in habit should be looked for and corrected, especially the usually faulty dietary habits. The use of laxatives is reprehensible in that it only prolongs the time when the patient can be cured of his bad habit. Temporary treatment for rectal conditions, especially fecal impaction, may be necessary.

#### HEMORRHAGE

Gross bleeding from the intestines may be due to ulceration, vascular damage or tissue necrosis. It may result from mucosal trauma, irritation, infection, radiation therapy, corrosive poisons, parasitic infestation or allergic reactions, from neoplasms, both benign and malignant, from vascular occlusion in the mesentery or in the intestinal wall as in intussusception or other intestinal obstruction, from simple mucosal bleeding as in blood dyscrasias, from anal and rectal lesions, and from diverticula, especially when inflamed. Practically all the conditions enumerated as causes of diarrhea may also cause bleeding. Familial telangiectasis may be overlooked if not remembered.

The hemorrhage may be slight, not sufficient to have an effect on the blood picture, or it may be severe enough to produce shock, acute anemia, anoxia and death. The bleeding may be so insidious that its only clinical manifestation is anemia, although as a rule occult blood will be found in the stools in such cases.

Clinical evidences of the *site of bleeding* may be entirely lacking or very deceptive. In general, bright red blood indicates bleeding from the distal colon, as from neoplasm, hemorrhoids or other anal sources. A tarry mixture, black in color and sticky in consistency, is characteristic of hemorrhage from the upper part of the alimentary canal, from the duodenum upward or even from the respiratory tract, and may be associated with hematemesis or hemoptysis. Bleeding from areas between these two extremes may be deceptive, although in general the darker and more altered the blood appears, the higher up is its source. Clots may form in

days to have a regular bowel movement every day or two and should be encouraged to keep up the various necessary measures

*Recent constipation* especially when associated with retrostaltic symptoms and perhaps alternating with diarrhea calls for a complete study as recommended above with especial attention to rectal examination including proctoscopy. A barium enema study should be made first and if no obstruction is found a complete gastrointestinal series should be carried out. Cancer should be borne in mind although this is associated more with diarrhea than constipation.

#### CONSTIPATION DUE TO ORGANIC DISEASE

Organic conditions which may interfere with normal bowel habit include the following

1 *Anorectal conditions* such as hemorrhoids fissures fistulas papillitis neoplasms and lymphogranuloma venereum are discussed under the chapter on the Rectum (p. 413). Though most of these conditions are associated at times with diarrhea spasm or actual obstruction due to them will occasion a retention of feces a fecal impaction and actual constipation. Liquified feces may later pass around the impaction or tunnel through it. The underlying cause should be treated or removed. In a patient with marked spasm and fecal impaction preliminary treatment is often necessary. Forcible dilatation of the sphincter if necessary under anesthesia will relieve a spasm. An impaction may be softened with oil from above and below or with repeated instillation of soapsuds or one of the newer enema preparations in disposable plastic bags. The impaction may then be broken up with the finger and delivered piecemeal perhaps aided by a speculum or forceps. With the rectum cleared the usual castor oil preparation for a careful proctoscopy and a barium enema x-ray can be given and further complete study carried out even though an obvious rectal lesion requires treatment. Any rectal lesion may be associated with serious lesions higher up. Hemorrhoids in particular may be caused by pressure on the hemorrhoidal arteries from cancer higher up or from pressure of an abdominal or pelvic tumor. In ulcerative colitis even though bloody rectal discharges are frequent impaction with actual constipation is not unusual.

2 Almost any upper gastrointestinal tract disease especially if associated with spasm and retrostaltic symptoms and accompanied by intake of little food and insufficient residue may be complicated by constipation. In treating any such disease it is well to remember that the diet should contain enough normal or added residue to take care of bowel function. If temporarily a nonresidue diet is prescribed mineral oil by mouth or rectum or one of the wetting agents is preferable to cleansing enemas to promote bowel movements. It is not necessary to have daily stools.

3 *Pelvic disease associated with enlargement of the uterus or adnexa or of the prostate* may cause sufficient pressure on the rectum or sigmoid

to interfere with bowel movement. The indications for treatment are usually obvious.

4 Some *endocrine dyscrasias* especially hypothyroidism and hyperparathyroidism are often associated with severe constipation and require appropriate treatment.

5 Actual obstructions of various degrees from bands, kinks, cicatricial stricture, diverticulitis, anomalies such as congenital megacolon and from cancer must be discovered and cared for appropriately.

#### SUMMARY

Constipation is a symptom and should never be treated *per se*. Before embarking on any line of treatment a complete study should be carried out in order to make sure that no organic disease especially cancer is being overlooked and its prompt care neglected. In the cases not due to organic disease errors in habit should be looked for and corrected especially the usually faulty dietary habits. The use of laxatives is reprehensible in that it only prolongs the time when the patient can be cured of his bad habit. Temporary treatment for rectal conditions especially fecal impaction may be necessary.

#### HEMORRHAGE

Gross bleeding from the intestines may be due to ulceration, vascular damage or tissue necrosis. It may result from mucosal trauma, irritation, infection, radiation therapy, corrosive poisons, parasitic infestation or allergic reactions from neoplasms both benign and malignant, from vascular occlusion in the mesentery or in the intestinal wall as in intussusception or other intestinal obstruction, from simple mucosal bleeding as in blood dyscrasias, from anal and rectal lesions and from diverticula especially when inflamed. Practically all the conditions enumerated as causes of diarrhea may also cause bleeding. Familial telangiectasis may be overlooked if not remembered.

The hemorrhage may be slight, not sufficient to have an effect on the blood picture, or it may be severe enough to produce shock, acute anemia, anoxia and death. The bleeding may be so insidious that its only clinical manifestation is anemia, although as a rule occult blood will be found in the stools in such cases.

Clinical evidences of the *site of bleeding* may be entirely lacking or very deceptive. In general bright red blood indicates bleeding from the distal colon as from neoplasm, hemorrhoids or other anal sources. A tarry mixture, black in color and sticky in consistency, is characteristic of hemorrhage from the upper part of the alimentary canal from the duodenum upward or even from the respiratory tract and may be associated with hematemesis or hemoptysis. Bleeding from areas between these two extremes may be deceptive although in general the darker and more altered the blood appears the higher up is its source. Clots may form in



any part of the tract where stasis permits of coagulation although in general this will be the rectum or sigmoid

*Admixtures with feces* indicate bleeding from above the point where stool formation takes place the descending colon and sigmoid. Below this point the blood may coat the stool or be passed independently of it. However in diarrheal stools this differentiation may not be detectable.

*Admixture with mucus* indicates irritation inflammation or infestation and may occur as a result of changes surrounding a neoplasm.

*Admixture with pus* occurs in pyogenic infections of the bowel wall as in dysenteries or in salmonella staphylococcal or pyocyanus infections the pus being green in the last named. Fistulas discharging into the lumen will also show pus in the stools or the pus may be passed independently.

### Occult Blood

As has been mentioned elsewhere the finding of occult blood is of little significance. Even when it is absolutely certain that the patient has taken no food containing blood for four days before the stool is tested occult blood may be due to slight bleeding anywhere in the alimentary canal respiratory system or mouth.

### Visible Blood

The finding of visible blood is always an indication for a complete physical examination including proctoscopy. Even when hemorrhoids are found to be bleeding it is important to rule out cancer of the sigmoid as a cause of the hemorrhoids. The public is being educated to recognize the importance of bleeding and nearly all patients are not only willing but also anxious to undergo a complete gastrointestinal study including thorough x rays to find the cause.

### Treatment

Bleeding should not be treated as such until its exact cause is known. However while a study is progressing or when the bleeding is known to originate principally from the mucosa of the distal colon treatment may be of some value.

*General Treatment* General treatment such as the use of vitamin K and calcium may be tried although it will be of real value only when coagulation factors have been found to be deficient. Diet and other measures suitable to the disease causing the hemorrhage are of course necessary. *Transfusion may be required to save life* but will not prevent further bleeding.

*Local Treatment* When hemorrhage is profuse it may be treated temporarily by (1) insufflation through the proctoscope of astringent powders containing equal parts of bismuth subnitrate thymol iodide and

tail which will adhere to the mucosa for a while. This treatment perhaps preceded by application of 10 per cent silver nitrate solution is of value in cases showing raw bleeding surfaces. (2) Instillation of 10 per cent hot aqueous gelatin solution 5 to 8 ounces at a temperature of 110 to 115° F to be run slowly through a heated funnel and catheter with the patient in the Sims position (left lateral with knees drawn up) and allowed to remain as long as possible. The heat and gelatin favor coagulation and soothe the mucosa. This can be done three or four times a day. The strength of the gelatin solution corresponds roughly to that used for making gelatin dessert or it can be used at double that strength. It jells slightly at body temperature. This treatment is sometimes of value when much bleeding occurs in the cases of rectal bleeding, ulcerative colitis and even temporarily in cancer. (3) Treatment of the cause of the bleeding may require medical or surgical measures.

Any one or a combination of the foregoing symptoms may be produced by diseases of the intestines. Their presence calls for a careful study including a history of the relations of the symptoms to function and careful physical examination and laboratory procedures. Under the general head of treatment the procedures recommended for each symptom particularly the diets will be further discussed.

### Physical Examination

In addition to a careful abdominal examination as described under symptoms a general physical examination is indicated in every case to determine whether there are diseases or other abnormalities elsewhere which might cause intestinal symptoms or might influence their treatment.

*Proctoscopy* is always indicated for we have seen what a great influence even mild rectal lesions have upon intestinal function. Early diagnosis of cancer within reach of a long proctoscope depends upon this examination. *Biopsy* or *cytological* studies may clinch a diagnosis. It is a grave mistake to perform proctoscopies only on patients who have definite rectal or sigmoid symptoms. For a description of proctoscopy see page 419.

### Laboratory Examinations

*Fractional gastric analysis* can at times give valuable information. Achylia may be the cause of gastrogenous diarrhea. A duodenal ulcer may be recognized (see p. 260).

### The Long Intestinal Tube

The Miller Abbott tube may be of diagnostic as well as therapeutic value. Continuous suction from the intestine proximal to an obstruction

may bring up bloody contents careful cytological study of which may disclose malignant cells parasites or ova

### Stool Examinations

These are always indicated and more than one examination is absolutely necessary The stools in diarrhea are described on page 317

**Gross Appearance** In describing a stool the following factors must be taken into consideration

**FORM AND CONSISTENCY** When *normal* these factors depend on diet fluid intake and exercise the stools should be formed 1 or 1½ inches in diameter in one or several pieces but they may be soft or mushy In *dehydration* and with *insufficient residue* in the diet small marbles or pellets are formed sometimes conglomerated In *diarrhea* the stools may be loose or watery In *rectal narrowing* due to intrinsic tumor or external pressure ribbon or pencil like stools occur

**COLOR** This varies with the type of food On an average diet it is brown Green vegetables give a greenish tint much milk a yellow color meat brown beets and carrots red some berries black Bismuth and iron cause black discoloration barium white Clay colored not yellowish stools indicate biliary obstruction

**ODOR** Normal characteristic stools are not too disagreeable in odor but are affected by the food ingested With a milk diet there may be no odor or a sour smell Putrefaction of proteins produces hydrogen sulfide and other putrid odors Ulcerating cancers have an extremely rotten pungent odor In some persons disagreeable odors come from the cabbage family onions and turnips or other ingested foods or drugs

**ADMIXTURES** *Mucus* as a clear jelly in cysts or membranes is caused by irritation *Pus* clear or mixed with feces comes from pyogenic infection as in bacillary dysentery or from abscesses discharging into the bowel *Blood* bright in color on the surface of the stool or on toilet paper comes from anorectal bleeding mixed with mucus from ulcerative colitis in small flecks of bloody mucus from amebic dysentery tarry sticky and black from bleeding high up in the small intestine or stomach *Occult blood tests* are mainly of value if negative indicating no bleeding lesion Occult blood reactions may come from ingested meat or meat extractives from three or four days before or from even slight bleeding from nose to anus *Worms* should be looked for Gallstones and other *foreign bodies* may be found on putting stools through a sieve

**Microscopic Examination** Microscopic search for parasites and ova may require eight to ten stools Meat fibers starch granules fat globules or fatty acids must be looked for after three or four days of a Schmidt diet (see under Pancreas p 584) Repeated cultures may disclose pathogens Identification of some organisms like salmonella and dysentery groups is often greatly helped by bacteriophage study *Cytological studies* may disclose malignant cells

## X-ray Examinations

Studies by *scout films* of the gas shadows and their arrangements may be of great help in deciding upon the presence and location of an obstructive lesion or may show free gas under the diaphragm as an indication of perforation.

The value of a complete gastrointestinal series following an opaque meal and a barium enema study cannot be overestimated. It is essential that such a complete x-ray study be done on every patient who has had symptoms longer than a week or two especially on older patients. It is never sufficient to do only a barium enema when a patient appears to have colonic symptoms or only a gastrointestinal series when symptoms suggest disease of the small intestine. Many grave errors in diagnosis are made in this way. Details in regard to x-ray studies will be found in the articles on the Stomach (p. 227) and the Colon (p. 357).

## Treatment

### Medical Therapy

It is best to avoid any treatment for the purpose of allaying symptoms until a complete diagnosis has been made. Many a cancer is overlooked until too late for cure because sedatives or antispasmodics have temporarily relieved symptoms making both patient and physician feel that a complete study may be unnecessary.

**Diet** While a study is being undertaken a well balanced diet should be prescribed. When there is no suspicion of an obstruction this should be the standard x-ray diet we have recommended the high residue diet discussed above under treatment of the symptoms of constipation. When there is a suspicion of partial obstruction a nonresidue diet should be prescribed. Our ulcer diet (p. 267) is suitable. This diet is also indicated whenever a nonresidue diet is indicated. It should be modified to avoid foods to which a patient may be allergic as described in the discussion of gastrointestinal allergy. The proper diet for each intestinal disease will be discussed later. For a general discussion of diets see opening chapter (p. 20).

**Immunotherapy** Vaccines and serums may be used in suitable cases especially for prophylaxis. Bacteriophages are of great value in certain specific infections such as bacillary dysentery, salmonellosis and infection with *Pseudomonas pyocyanea* or *aeruginosa* formerly called *Bacillus pyocyaneus*.

**Medicinal Therapy** It is wise to use as few drugs as possible because of the danger of drug allergy. A few of the agents which may be indicated are as follows:

1. **Soothing demulcent drugs** including bismuth, kaolin, pectin, agar or psyllium derivatives. The first three are useful alone or in combination for diarrheas, the last two for constipation although also useful at times for diarrhea.

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emergency an ample incision should be made to enable careful and complete exploration

*Preparation for Operation* The preoperative care of the patient is perhaps the most important measure to insure a good result. Its purpose is to get the patient into the best possible condition to withstand the operation. Plenty of time preferably at least two or three weeks should be given to preparation except in emergencies.

In an *acute emergency* about all that can be done is to combat shock and dehydration while the patient is being prepared. Parenteral injections of stimulants, fluids, electrolytes, vitamins and blood are of tremendous value. Sedation should be kept at a minimum. Instrumentation should be avoided as much as possible, although gastric aspiration and intestinal decompression by means of a Miller Abbott tube are justifiable in obstruction when no fear of perforation is present. A cleansing enema or irrigation is at times desirable in colonic obstruction but should be carefully performed.

When time permits even in cancer adequate preparation may spell the difference between life and death. The means suggested for preoperative care in operations on the stomach (p. 232) are equally important in intestinal operations. Postoperative care should also follow the same general outline.

## Anomalies and Diseases of the Intestines

### ANOMALIES OF THE INTESTINES

#### ANOMALIES OF THE SMALL INTESTINE

Aside from the serious anomaly of duodenal atresia, anomalies of the small intestines are due to faulty rotation or faulty development of the peritoneum or mesentery. In the cases of *faulty rotation* the location of the small intestine is altered with a tendency to be on the right side of the abdomen. Almost the entire small intestine may at times be found to the right of the ascending colon and unless thought of will cause confusion on x-ray study. This anomaly as well as anomalous peritoneal folds and fossas, bands and adhesions may favor the development of *internal hernias*. Mesenteric anomalies such as long or short mesenteries or gaps in the mesocolon or mesentery may also be the cause of such hernias. Insufficient fusion of the mesentery may permit the development of a volvulus. The duodenum may show more or less redundancy and at times there are congenital adhesions at the duodenojejunal angle. Bands or membranes may cause arterio-mesenteric occlusion. Congenital cysts of the duodenum and an annular pancreas surrounding the duodenum are often difficult to recognize being mistaken for ulcer. Ileocecal valve prolapse, the invagination of loose ileal mucosa through the valve into the cecum may resemble ileal polyp with intussusception.



2 *Antispasmodic and anticholinergic drugs* such as atropine and belladonna and the newer ones which are constantly being produced are of limited value

3 *Analgesics* including aspirin and various old and new coal tar preparations may be used to control pain in inoperable cases until they must be replaced by narcotics

4 Of the *narcotics* opium derivatives morphine and Demerol are mostly used although tincture or extract of opium is often helpful

5 *Sedatives* are much too extensively used and in themselves often cause gastrointestinal symptoms Phenobarbital is as good as any of the barbiturates given in doses of  $\frac{1}{2}$  grain to 2 or 3 grains according to need usually for sleeplessness or great apprehension Chloral is still extensively used as are bromides The new tranquilizers may cause intestinal symptoms and must be used with caution

6 *Intestinal antiseptics or bacteriostatic drugs* have long been used The *sulfonamides* were formerly much used but the soluble ones cause great damage especially to the renal tubules For colonic infections the insoluble sulfonamides sulfaguanidine and Sulfasuxidine are at times of value even today and are used by many surgeons in preparation for colonic surgery *Antibiotics* have been prescribed to excess Though they cause a rapid reduction in the bacterial count of the stools some bacteria may become resistant to them with resulting serious infections such as staphylococcus infection and pseudomembranous or hemorrhagic enterocolitis At other times destruction of bacteria may cause an increase of fungi in the bowel especially monilia which may cause persistent diarrheas Allergy to the antibiotics may also result in severe diarrheas even ulcerative colitis Some of the newer antibiotics notably tetracycline and oleandomycin appear to have fewer bad sequelae but should be used with caution Mycostatin often clears up a monilial infection promptly and has been used in the prophylaxis of this infection by prescribing it with antibiotics

7 *Laxatives and cathartics* should rarely be used in chronic cases For a discussion of their use as well as of enemas the reader is referred to page 44

### Surgical Treatment

Operation is often necessary but usually only for neoplasms and for complications of other diseases such as obstruction or perforation For abscesses incision and drainage may be required In obstruction external openings such as enterostomies and colostomies may be made either for permanent use in inoperable cases or preliminary to an attempt at a curative resection Although resection of the entire colon can be done without too much risk removal of more than one third of the small intestine is usually not compatible with life At any operation except in

tion will produce the symptoms of obstruction from any cause which occasionally may be suggested by the previous history

**Physical Examination** A thorough general physical examination may reveal anomalies elsewhere in the body. As anomalies are not usually confined to one region suspicion should be aroused that there may be an abnormality in the abdomen accounting for gastrointestinal symptoms. Abdominal examination may show nothing abnormal although at times in thin patients the abnormal arrangement of the gas filled intestine may be suggestive especially when coils of small intestine are felt far to the right. This is more marked in intestinal obstruction. The findings in intestinal obstruction are discussed elsewhere (p. 67)

**Diagnosis** X-ray examination will usually reveal the diagnosis. It is necessary however to keep in mind that anomalies can occur so that they may not be confused with other diseases.

**Treatment** As long as anomalies are causing no symptoms they require no treatment when accidentally discovered. Even when symptoms occur if their cause can be discovered and postural changes relieve them operation should be deferred. Postoperative adhesions and kinks may result from attempting to change the position of the intestine or from freeing large adhesions. When obstruction occurs intubation and suction are indicated and operation may be necessary at times requiring resections. Duodenal atresia is usually inoperable. Complete colonic atresia is not compatible with life.

**Prognosis** Most anomalies do not interfere with a normal existence. Others especially when associated with important anomalies elsewhere lower a patient's general resistance and may cause trouble at any time by favoring the development of disease such as ulcer or cancer.

### Megacolon

Megacolon is a condition in which the colon is dilated and elongated and the colonic wall is hypertrophied. The dilatation usually involves only the proximal part of the colon and ends abruptly with narrowing distal to it. The dilatation may be moderate or extreme some cases having been reported up to 14 inches in diameter. The dilated portion is also lengthened sometimes to double the normal length. With the stretching weak areas with diverticulation may occur. The dilated colon has at times been found to hold as much as 30 to 50 pounds of feces. The weight may stretch the mesocolon so that the colon becomes hypermobile.

**Pathology** Primary and secondary forms of megacolon are usually described although at times it may be difficult to differentiate between them.

Primary or congenital megacolon also called Hirschsprung's disease is found mostly in male infants and children and even in the fetus. In some cases the condition is found in adults even up to eighty years of age. It

## ANOMALIES OF THE LARGE INTESTINE

The colon may show atresias varying in degree from total or partial absence of lumen in parts of the colon to a complete atresia of the entire colon which then appears as a fibrous cord. Duplication and triplication of the colon have also been seen. Redundant flexures or double flexures are not uncommon. Hypermobility of the cecum is not unusual. Anomalies of position of the colon are associated with various degrees of nonrotation or partial nonrotation and its corollary, varying degrees of descent of the cecum. Both the ascending and descending colon may lie on the left side in double barrelled fashion with the small intestine on the right side as described above. The cecum may lie in the right upper quadrant or anywhere between there and deep in the pelvis. Because of this the appendix may be found in unusual locations making diagnosis of acute appendicitis difficult. Inflammation of an abnormally high appendix has been known to simulate cholecystitis and cases are reported of perforation through the diaphragm. Various forms of fixation and congenital bands and adhesions are also encountered. Congenital megacolon and diverticulosis are described following this general discussion of anomalies.

A rare intestinal anomaly that must be borne in mind to avoid confusion when it is encountered is *transposition of viscera*. Attention to this condition is alerted by the presence of dextrocardia with which it is usually associated. Left sided liver, gallbladder and appendix are the most spectacular findings although the peculiar position of the stomach with the pylorus to the left may cause initial confusion. *Congenital herniation* or eventration of the diaphragm may contain intestine as well as stomach and is discussed elsewhere.

*Meckel's diverticulum*, a vestigial remnant, is discussed under Diverticulosis.

*Pancreatic rests* may occur in the intestines and may simulate ulcers or cancers. They are discussed in the chapter on the Pancreas (p. 588).

*Complications*. As has been mentioned above the most important and troublesome complications are due to obstruction which may be complete or incomplete and intermittent or continuous. Obstruction may be caused by kinks, internal hernia, volvulus or external compression.

*Symptoms*. Anomalies having been present since birth the intestine tends to adapt itself to them and no symptoms may result unless complications develop. In some cases symptoms may be caused by kinking or partial occlusion which occur when the body is in a certain position and may be relieved when the patient changes his position. For instance such symptoms as distention, rumbling, difficulty in expelling flatus, cramps, nausea and even vomiting may be relieved by a patient's lying on one side or the other, standing, stooping, bending or rubbing his abdomen. A history of such a sequence of events should occasion suspicion of an anomaly or of postinflammation or postoperative adhesions. Real obstruc-

and more or less atrophied and shows little or no peristalsis. This has been attributed to congenital absence of the myenteric ganglia (Auerbach's plexus) which are essential to peristalsis. The absence of peristalsis acts like an obstruction causing a backing up and dilatation above. It has also been assumed that a congenital or acquired narrowing of the rectosigmoid may be the cause. In cases in which the rectum is also dilated the narrowing has been assumed to be in the anal region. Congenital atresia, bands, kinks, a long sigmoid or other anomalies have also been postulated as causes. Overactivity or underactivity of the sympathetic, underactivity of the autonomic innervation and segmental nerve pressure have been suggested. The resemblance of megacolon to megaoesophagus has suggested that the cause of each may be similar; that achalasia of the anal sphincter may be the exciting cause of megacolon as achalasia of the cardia is of megaoesophagus.

*Secondary* or acquired megacolon has been attributed to both organic and functional causes. *Organic causes* would include narrowing of a segment of bowel producing dilatation behind it. This narrowing may be due to any chronic inflammatory disease, allergic reaction, neoplasm, adhesions, cicatricial contractions, torsion, operative procedure or other condition capable of reducing the bowel lumen. It is a question whether the term "megacolon" should be used for such a condition. It is usually described merely as distention due to partial or complete obstruction.

*Functional* or acquired megacolon of varying degree has been ascribed to so-called chronic habitual constipation, a failure to observe the natural rules relating to the bowel habit discussed on page 320. Gradually the colon becomes increasingly dilated, impactions occur and obstruction may be simulated or actually supervene.

*Symptoms.* Early symptoms occurring at birth or soon afterward consist in abdominal distention and vomiting, occasionally fecal. There may be diarrhea at first, then constipation, very severe. Later in childhood general malnutrition and anemia, with physical and mental retardation in a male child, should occasion a suspicion of Hirschsprung's disease. Occasionally diarrhea may occur due to liquid feces passing alongside the impaction or tunneling through it, so that constipation and diarrhea may alternate. In some children the parents simply persist in giving cathartics and enemas until complications occur. These may include paralytic ileus and injury to the bowel from overdistention, causing enterocolitis, ulceration or perforation. Occasionally a volvulus may occur. Pressure upward on the diaphragm with resulting pneumonia may be a cause of death. The mortality in presence of complications is high. Patients with mild cases in which the narrowed segment is not too long to permit pressure from above to push bowel content through it may survive to late childhood or even adult life. In such cases the distention is usually not accompanied by pain and is often ignored. Upward pressure against the

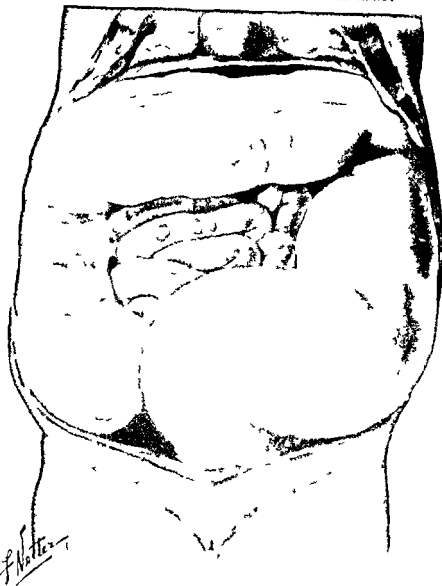


Figure 55 Megacolon Enormous distention of colon especially the sigmoid (From the Ciba Collection of Medical Illustrations by Frank H Netter M.D. Copyright Ciba Pharmaceutical Products Inc.)

has usually been called idiopathic. This might conceivably also apply to the rare type with narrowing involving the entire colon including the rectum often called *microcolon*. Usually there is a dilatation of the proximal colon most frequently extending down to the descending colon sigmoid or even upper rectum where it ends abruptly. Below this there is a segment of varying length occasionally extending as far as the anal canal which shows a narrowing of varying degree. This segment is atonic

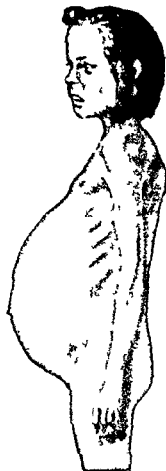
even through an overcoat. The chest may show dilatation with intercostal spaces stretched and occasional bulging of the left lateral wall. The stretched abdominal skin may be atrophied and shiny. Distasis recti may make intestinal peristalsis clearly visible. Superficial veins may be prominent. Palpation may disclose movable masses of hard feces, and the abdomen and chest are usually tympanic. Rectal digital examination may show a spastic or abnormally relaxed sphincter or may locate a neoplasm or pressure from a large uterus or prostate. The rectum may be packed with feces. In general the patient shows evidence of generally decreased tissue fit and poorly developed musculature, especially of the legs.

*Proctoscopy* is usually easy, the instrument passing freely through the relaxed and often dilated rectum well into the sigmoid until the narrowing is encountered. Impacted feces may have to be removed before a satisfactory examination can be carried out.

*X-ray Examinations* A scout film will show loops of bowel distended with gas, but differential diagnosis is often difficult. A *barium enema* study must be done with great caution, lest the barium become trapped in the colon and be difficult to remove. The distended bowel, unless filled solid with feces, will hold tremendous quantities of the barium mixture, 2 to 3 quarts often being required to fill only the sigmoid, which may be seen extending up to the diaphragm. If the whole colon is filled, its



Fig. 57. Congenital megacolon. General colonic distention, especially of sigmoid. Note narrowing below. (Feldman: *Clinical Roentgenology of the Digestive Tract*, 3rd ed. Williams & Wilkins.)



*Figure 56* Congenital megacolon. Marked abdominal distention and general emaciation. (From the Ciba Collection of Medical Illustrations by Frank H. Netter, M.D. Copyright Ciba Pharmaceutical Products, Inc.)

diaphragm causing embarrassment to the heart and lungs may result in the patients being treated for supposed trouble above the diaphragm. Headaches, dizziness, anorexia and nausea may occur after long delays in defecation. There may be little change in weight except in severe cases. Finally, after long continued inability to move the bowels with no stool for weeks unless strenuous measures are adopted including large doses of cathartics and frequent enemas together with vomiting, distention, excessive borborygmi, abdominal pains and more or less severe obstructive symptoms, the patient may at length call for an x-ray study which will disclose the state of affairs. With development of these complications the patient becomes a serious problem.

*Physical Examination* The abdominal distention is the most noticeable feature and may be so excessive in severe cases as to be noticeable

times daily has been advised but in addition to being of only temporary advantage these agents may produce unfortunate side effects. Occasional marked distention may require temporary decompression by means of the Miller Abbott tube from above or a rectal tube from below. In rectal impaction the measures discussed on page 441 may give relief. In some patients daily (or nightly) oil retention enemas of a few ounces of oil may need to be used for long periods of time (see p. 46) but may be reduced in quantity and frequency as time goes on. In impactions in the proximal colon kneading even under anesthesia may be tried to soften them and push them to the distal colon in order to be within reach of local treatments. Dilating the anal sphincter and the passage of bougies through a narrowed segment may be attempted. Upon failure of all these measures operation must be considered.

Surgical treatment is often indicated in early infancy especially when obstructive symptoms occur. It is of course definitely indicated in most forms of organic obstruction. In congenital megacolon various types of operation were performed in the past such as ileosigmoidostomy, removal of the dilated colon, nerve blocks of the sympathetic and parasympathetic nerve ganglia and operations for complications such as volvulus. In recent years the surgical attack has been in the lower adynamic segment at first by removal of parts of the sigmoid. The most recent procedure the "pull through operation" first devised by Swenson is being rather generally adopted. The purpose is to remove if possible all the diseased distal segment down to the anal orifice and to pull down the uninvolved and dilated colon from above suturing it to the anal canal in such a way as to establish continuity of the bowel and preserve sphincter function. The initial mortality rate is high and complications due to severance of nerves controlling defecation and urination may result in permanent disability. To obviate this damage a newer operation has been tried in which only the mucosa of the involved portion is removed the uninvolved portion being pulled through this muscular tube and sutured to the anus.

*Prognosis.* Congenital megacolon is a serious disease. In early infancy without operation the mortality rate has been reported as from 50 to 90 per cent. With the newer operations the rate has been reduced to 30 or 40 per cent or even lower but complications are frequent. In the mild cases with not too large a segment involved medical treatment is usually satisfactory and such operation when required may be expected to produce better results.

### Diverticulosis

Diverticula are not uncommon and they occur in both the large and small intestine. They are found mainly in the second portion of the duodenum where they are more often single than multiple. The first portion is rarely the seat of true diverticula but false diverticula the



individual parts will be difficult to recognize unless careful fluoroscopy has been done during the filling process. The dilated colon shows few if any haustrations. *Barium orally administered* may go along normally until it "gets lost" in the distended colon producing only a mottled appearance when mixed with the feces.

In severe cases a *chest x ray* will show the distended colon pushing upward the elevated left dome of the diaphragm, compression of the lungs and displacement of the heart to the right.

No *laboratory tests* are of any particular value unless complications develop or specific infections or neoplasms may be recognized by culture or biopsy.

**Diagnosis** The obstinate constipation and abdominal distention together with the *x ray* findings will determine the diagnosis. An organic cause must be looked for in the rectum, sigmoid and pelvis. In the congenital type biopsies of the narrowed segment have been suggested to determine absence of myenteric ganglia, but the specimens must be obtained from so deep in the colonic musculature that perforation is to be feared. Even specimens obtained at operation and examined by frozen section often do not show this defect. To be ruled out are the "potbelly" of rickets with its skeletal changes, tuberculous peritonitis with its peritoneal fluid and chest findings, and large abdominal cysts which are of course filled with fluid. Ascites while producing a protruding abdomen cannot be mistaken for gas.

**Complications** Though infections in these patients are serious they seldom occur. Fecal impaction may be a distressing occurrence for both patient and doctor. Volvulus of the sigmoid is fortunately rare. A complete volvulus is probably a fatal complication. It is frequently overlooked and operation is dangerous. The possibility of a real intestinal obstruction must always be borne in mind. The other complications mentioned under *Symptoms* must be carefully watched for.

**Treatment** In so called acquired or secondary organic megacolon the treatment is that of the original organic condition and is discussed under the various diseases mentioned as causes, obstruction. A complete diagnosis should be made if possible before operation is attempted and adequate preparation for surgery must be carried out as described on page 232. In acute emergencies the plan outlined under *Acute Conditions of the Abdomen* (p. 59) is indicated.

In *congenital megacolon* the treatment must be guided by the severity of the condition. In mild cases with not too large a segment aperistaltic the treatment suggested for chronic constipation (p. 320) is usually successful. However there may be some abnormal distention always present aggravated by neglect of the diet and other treatment. The use of drugs stimulating peristalsis such as neostigmine methylsulfate 0.25 to 1.00 mg. parenterally or Urecholine 10 to 30 mg. orally two or three

times daily has been advised but in addition to being of only temporary advantage these agents may produce unfortunate side effects. Occasional marked distention may require temporary decompression by means of the Miller Abbott tube from above or a rectal tube from below. In rectal impaction the measures discussed on page 141 may give relief. In some patients daily (or nightly) oil retention en mas of a few ounces of oil may need to be used for long periods of time (see p. 46) but may be reduced in quantity and frequency as time goes on. In impactions in the proximal colon kneading even under anesthesia may be tried to soften them and push them to the distal colon in order to be within reach of local treatments. Dilating the anal sphincter and the passage of bougies through a narrowed segment may be attempted. Upon failure of all these measures operation must be considered.

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**Prognosis.** Congenital megacolon is a serious disease. In early infancy without operation the mortality rate has been reported as from 80 to 90 per cent. With the newer operations the rate has been reduced to 30 or 40 per cent or even lower but complications are frequent. In the mild cases with not too large a segment involved medical treatment is usually satisfactory and such operation when required may be expected to produce better results.

### Diverticulosis

Diverticula are not uncommon and they occur in both the large and small intestine. They are found mainly in the second portion of the duodenum where they are more often single than multiple. The first portion is rarely the seat of true diverticula but false diverticula the

result of walled off perforated ulcers are found there frequently. Diverticula of the remainder of the small intestine from the second portion of the duodenum down constitute about 25 per cent of gastrointestinal diverticula. They are often multiple but rarely in large numbers. They are found in the duodenum three times as frequently as in the jejunum and ten times as frequently as in the ileum. Over 70 per cent are in the colon. In many instances diverticula are found in two or more locations at the same time as in the esophagus and stomach as well as in the intestines.

**Pathology** A true diverticulum is a pocket or pouch protruding like a hernia from the intestine and communicating with its lumen at a weak point in the wall. It is always lined with mucosa and most frequently has all the coats of the intestine. Diverticula may vary in size from  $\frac{1}{8}$  to 4 inches or more in diameter the largest ones usually having no muscular coat. Complications are rare. With inflammation ulceration or trauma the diverticula may bleed or occasionally may perforate causing either peritonitis or peridiverticulitis. Rarely they may perforate into neighboring organs producing fistulas to the intestine or gallbladder. Small intestinal diverticula may contain pancreatic rests which may cause ulceration hemorrhage and perforation.

**Etiology** The cause is unknown some considering them congenital others that they are due to localized areas of weakness either congenital or produced by diseases such as enteritis or colitis or by excessive use of cathartics or enemas.

**Symptoms** Although some of the larger diverticula look formidable they rarely cause symptoms unless complicated. Symptoms which were attributed to small intestinal diverticula such as epigastric distress or pain belching regurgitation and vomiting have continued after successful removal of diverticula. Complications such as inflammation ulcer perforation fistulation and obstruction are most uncommon and if they occur will produce symptoms not directly referable to the diverticula. In the large intestine also no symptoms occur except with complications.

**Physical Examination** Unless complications are present the examination will disclose nothing abnormal. There are also no laboratory or mechanical aids to diagnosis except the x-ray. Symptomless diverticula are usually discovered on routine x-ray study. Proctoscopy rarely shows the opening of a diverticulum.

**X-ray Examination** With careful and repeated barium meal study and films taken at one half to one hour intervals upper small intestinal diverticula can usually be demonstrated if present (Fig 37 b p 242). Diverticula in the second and third portions of the duodenum may be covered with the barium filled stomach and may appear as deep ulcers unless films are taken in different positions in the lateral and standing positions especially. Diverticula in the remainder of the small intestine are frequently overlooked unless carefully searched for. Their shape

which is round oval or flasklike and their changes in location should attract attention although large ones are often mistaken for dilated intestinal loops After the barium meal has passed some barium may be retained later gas filled diverticula may be recognized Fistulas and other complications are rarely demonstrable

*Colonic diverticula* are best recognized in films taken forty eight hours or more after a barium meal when the characteristic multiple pockets of various sizes usually remain filled after the colon has emptied itself (Fig 58 *a*) Barium enema studies are unsatisfactory Deep haustration may be mistaken for diverticula on the other hand diverticula in their usual location on the posterior wall may be hidden by the filled colon (Fig 58 *b*) Even films taken after evacuation are not conclusive since the barium mixture may not enter the pockets before it has been expelled from the colon However much irritability in the region usually the sigmoid where diverticula have been seen with the barium meal technique indicates inflammation In some such cases diverticula can be seen to communicate with extravasations of barium a short distance away indicating walled off perforations

*Treatment* No specific treatment is required except for complications Operations for asymptomatic diverticula are notoriously unsatisfactory

*DIET* No special diet is necessary although it has been suggested that foods containing seeds should be avoided As seeds have practically never been implicated in complications this restriction seems superfluous In diverticulosis of the small intestines a normal well balanced diet or any diet indicated for other conditions found will be satisfactory In colonic diverticulosis I prefer a diet with plenty of residue such as my anti constipation diet (p 323) the theory being that well formed feces will

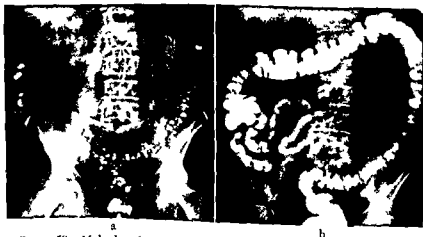


Fig 58 Multiple colonic diverticula from same patient *a* Forty eight hours after barium meal—diverticula from cecum to sigmoid *b* Barium enema only—a few diverticula seen in the descending colon and sigmoid

result of walled off perforated ulcers are found there frequently. Diverticula of the remainder of the small intestine from the second portion of the duodenum down constitute about 25 per cent of gastrointestinal diverticula. They are often multiple but rarely in large numbers. They are found in the duodenum three times as frequently as in the jejunum and ten times as frequently as in the ileum. Over 70 per cent are in the colon. In many instances diverticula are found in two or more locations at the same time as in the esophagus and stomach as well as in the intestines.

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**Etiology** The cause is unknown, some considering them congenital, others that they are due to localized areas of weakness, either congenital or produced by diseases such as enteritis or colitis, or by excessive use of cathartics or enemata.

**Symptoms** Although some of the larger diverticula look formidable, they rarely cause symptoms unless complicated. Symptoms which were attributed to small intestinal diverticula, such as epigastric distress or pain, belching, regurgitation and vomiting, have continued after successful removal of diverticula. Complications such as inflammation, ulcer, perforation, fistulation and obstruction are most uncommon and if they occur will produce symptoms not directly referable to the diverticula. In the large intestine also no symptoms occur except with complications.

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rapidly either spontaneously or as a result of treatment. It may be very acute with considerable surrounding exudate or perforation may occur. This may produce general peritonitis in "acute abdomen" or may be walled off producing peridiverticulitis and forming a large mass. Pyogenic infection with abscess formation may ensue and infection may be carried through the portal vein to the liver producing abscesses or to the kidney region producing perinephritic abscess. Perforation rarely takes place into the bladder or ureter causing urine to be contaminated with feces. *Chronic diverticulitis* may present a mass for months before subsiding or may have episodes of acute inflammation.

**Symptoms** Pain in the region of the lesion is the usual finding. In sigmoid diverticulitis pain occurs in the left lower quadrant. Depending upon the severity of the inflammation it may be mild or severe, constant or colicky, may act like a "left sided appendicitis" or with perforation like an "acute abdomen." It is usually accompanied by constipation although diarrhea is frequently found occasionally with bleeding (see p 315). Cecal diverticulitis causes symptoms like those of appendicitis and often is first recognized at operation. Fever varies. In chronic cases there may be a chronic diarrhea with more or less bleeding and recurrent attacks of acute symptoms.

**Examination** Tenderness is usually elicited over the site of the inflammation with rigidity dependent upon severity of the attack. With considerable peridiverticular infiltration a distinct firm mass is palpable, it is usually tender with distention above and is often mistaken for cancer. Leukocytosis and increased sedimentation rate are usually found.

**Proctoscopy** must be performed with caution and will usually show nothing abnormal. Rarely can a diverticulum be seen. I once had a patient whose inflamed diverticulum perforated as a result of the traction from a proctoscopy.

**X ray Examination** This must also be carefully done. In a patient with previously known diverticula a barium meal will not be required. The barium enema must be slowly trickled in watching for filling of diverticula and possible extravasation through a perforation as mentioned above (Fig 60). Mild cases will show more or less marked irritability and spasm in the region of the diverticula. Too much pressure may extend the extravasation and cause generalized peritonitis.

**Diagnosis** The diagnosis may be difficult. If the findings are typical it is fairly easy. Sigmoid cancer may be fairly well ruled out by the history, general condition and usual absence of fever and leukocytosis (although I have seen this in cancer also). If within reach of a proctoscope biopsy will settle the question. The x ray showing diverticula may also disclose an annular carcinoma although the two do not often go together. Tuberculosis occurs more frequently in the ileocecal region. Appendicitis may occur with inflammation or perforation of a cecal di-

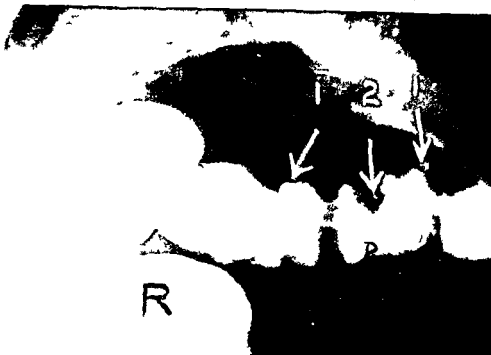


Figure 59 Sigmoid polyp in patient with diverticula 1 Diverticula (many more were found above) 2 polyp found at operation R rectum

not enter the small orifices of the diverticula but will pass on without irritation. This type of diet has been much more satisfactory than non-residue diets with which laxatives or enemas are often required. A liquid stool or enemal fluid under pressure can enter the diverticula and produce infection and even perforation.

**MEDICATION** Medication is rarely required. The diet will usually soothe the mucosa and allay any mild irritability. Mineral oil in  $\frac{1}{2}$  ounce doses or warm oil retention enemas 4 to 6 ounces at bedtime will cause no trouble.

**Complications** Diverticula of the small intestines rarely develop complications. With inflammation or perforation an acute abdomen may be simulated and then usually requires operation. Fistulation to neighboring organs also requires surgical attention. Fistulas to the pelvis will usually be mistaken for pelvic inflammatory disease. *Colonic complications* are based mainly on infection called colonic diverticulitis. Malignant degeneration has been mentioned but probably occurs in a pre-existing polyp (see Fig. 59).

### Colonic Diverticulitis

Colonic diverticulitis occurs more frequently in older patients averaging sixty years. It is usually in the sigmoid may be mild and subside

occurs from the umbilicus. With obliteration of the intestinal end a blind umbilical fistula is the result. Usually the entire duct is obliterated and it then becomes a mere fibrous cord. With the obliteration of the umbilical end and persistence of the intestinal ends the typical Meckel's diverticulum occurs. It is generally from 1 to 3 inches long but may be much longer. It usually enters the ileum about 3 or 3½ inches from the ileocecal valve but wide variations may occur in this respect also. Its lumen is slightly smaller than that of the ileum and its mucosal lining resembles that of the ileum. Occasionally aberrant pancreatic tissue may be found.

*Complications* or anomalies usually call attention to the presence of the diverticulum. If lined with gastric mucosa or if pancreatic tissue is present ulceration, hemorrhage or perforation may develop. Neoplasms both benign and malignant have been known to occur. Cancer is probably almost always due to an adenomatous polyp. If it has become detached from its umbilical end a diverticulum may induce an intussusception or may appear in an inguinal or femoral hernial sac called Littre's hernia. If the loose end when still attached to the umbilicus becomes adherent elsewhere as to the intestine, abdominal wall or bladder an internal hernia may be produced by the resulting band. A foreign body such as a tooth pick may enter and cause inflammation or perforation. Acute inflammation from secondary infection may cause intestinal obstruction, hemorrhage or perforation.

*Symptoms* In the absence of complications usually no symptoms are present although distention with intestinal content may at times cause discomfort due to interference with ileal peristalsis. Acute inflammation will produce symptoms like those of acute appendicitis. Obstruction and perforation will cause the usual symptoms whatever the cause. Hemorrhage when slight may not be noticed when massive it will resemble that from other causes, the stools being either tarry or bright red.

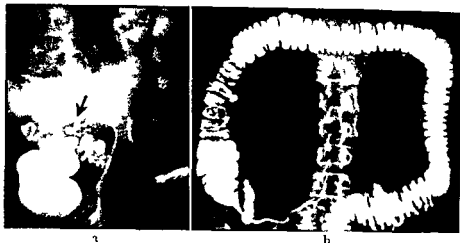
*Physical Examination* It is only with complications that abdominal examination will be of value. Perforation and hemorrhage will give the usual findings not diagnostic for any particular cause unless the history is suggestive. Acute inflammation will usually be mistaken for acute appendicitis. Occasionally a suggestive midabdominal mass may be palpable. A Littre's hernia may occasionally be identified.

*X-ray Examination* Not many years ago a Meckel's diverticulum had never been recognized by x-ray. Now with careful study of frequent films such a diverticulum can occasionally be recognized although it is often confused with a loop of intestine (Fig. 61).

*Diagnosis* As over 70 per cent of these diverticula are discovered before the age of thirty they should always be borne in mind when a youthful patient presents symptoms of acute appendicitis, small intestinal obstruction, unexplained intestinal hemorrhage and a large hernia. Exploration is usually required to make a diagnosis.

*Treatment* Surgery is the only treatment. At any operation it is always





*Figure 60* Colonic diverticulitis *a* Perforated sigmoid diverticulum Arrow points to extravasated barium *b* Barium enema study Note multiple diverticula and pathological appendix with sigmoid irritability

diverticulum and both require operation. Ulcerative colitis may be confused but may also occur with diverticulitis. Perforation into other organs will produce symptoms there which may be confusing unless a careful study is made.

**Treatment** It is always safest to have a surgical consultation early although operation is rarely advisable except for chronic obstruction due to fibrosis or adhesions with angulation for recurrent episodes or for a fulminating peritonitis. Resection may be required. In the mild or moderately severe acute case bed rest, parenteral feedings later followed by a nonresidue diet, rectal instillations of warm mineral oil 4 to 6 ounces and sulfonamides or antibiotics will usually give a good result after four or five days. X-ray therapy may be of benefit to reduce excessive induration. Gradual additions are made to the diet and mineral oil in 1/2 ounce doses per day may be started in two or three days but active catharsis or enemas are to be strictly avoided. After subsidence treatment as recommended for uncomplicated diverticulosis is carried out. The patient should be instructed to watch for pain, bleeding or changes in bowel habit.

**Prognosis** Most cases of acute diverticulitis unless perforated at the onset will subside with the treatment outlined. However an indication for surgery may arise so that a surgical opinion is of great value.

### Meckel's Diverticulum

Formerly thought to be extremely rare this vestigial remnant due to failure of obliteration of the vitelline duct in whole or part is now more frequently recognized and is estimated to be present in at least 2 per cent of all people twice as frequently in males as in females. With the entire duct from the umbilicus to the ileum remaining patent fecal discharge

other causes. The diagnosis depends upon careful small intestinal x-ray study. The treatment is surgical.

*Acute enteritis* practically never occurs alone. It is usually a part of a generalized inflammation of the stomach and intestines discussed under the subject of acute gastroenterocolitis and food poisoning (see p. 71).

*Chronic enteritis* is in the same category. It may follow acute episodes either as a result of continued infection or due to organic changes caused by the acute infection. More or less colitis also accompanies it. Mild forms unless accompanied by diarrhea are usually not recognized. Severe chronic enteritis, the regional form which also may involve the colon and ulcerative colitis which may involve the ileum are described separately. All these chronic diseases may be due to various causes including infection, infestation, allergy, x-ray burns and the other conditions which cause the severe forms except that the degree of reaction is less in the milder cases. Treatment is the same but results should be looked for much sooner in the mild cases.

#### REGIONAL ENTERITIS

Although it had been described previously, this disease was first described as an entity by Crohn in 1932 being called terminal ileitis. As its tendency to attack other parts of the small intestines and even the colon was recognized it became known successively as regional ileitis, then regional enteritis or cicatrizing enteritis. Now its colonic counterpart is called regional colitis. Although volumes have been written about it, this disease is really a rare condition. Previous to 1932 the findings in the small intestines were usually discovered at operation for appendicitis, were considered due to this disease or to tuberculosis and nothing was done about them. The infrequent incidence of this condition and its failure to appear in mortality statistics probably accounted for the general lack of interest in the disease. Although it may occur at any age, it is primarily a disease of young adults.

#### Pathology

The confusion about the pathological changes has been due to the various descriptions emanating from clinicians and pathologists who have described different stages of this disease. The first or acute stage shows inflammation, hyperemia, edema, increase in goblet cells, interstitial hemorrhages with some shallow ulcerations and occasional bleeding. These changes correspond to those found in any allergic mucosal reaction (see Allergy, p. 57). The interstitial spaces become clogged as a result of lymphatic endothelial proliferation which spreads by way of the lymphatics with development of lymphatic obstruction. Increasing edema and interference with mucosal circulation cause deeper ulceration. Lymphatic and fibrotic hyperplasia cause tubercle like or granulomatous



Figure 61 Littre's hernia 1 Rotated cecum 2 small Meckel's diverticulum 3 ileum in scrotum

wise to make a careful examination of the ileum especially when the operation has failed to show any obvious cause for symptoms. Removal of the diverticulum is usually not difficult and the results are excellent.

#### ENTERITIS AND COLITIS

*Duodenal ulcer and duodenitis* have been discussed with gastric ulcer and gastritis because of the fact that the first portion of the duodenum is really more a part of the stomach than of the remainder of the small intestine (see p. 214).

Periduodenitis, deforming the duodenal cap, is usually due to biliary tract diseases with which it is discussed (p. 234).

*Jejunal ulcer* has been mentioned as a complication of gastroenterostomy and its treatment has been discussed (p. 277). Primary jejunal ulcers occur rarely; ileal ulcers are most unusual. They are usually single, punched out, with a tendency to perforate either freely into another part of the small intestine or into the colon. The symptoms of simple ulcer may resemble those of peptic ulcer but with radiation to the left upper quadrant. Hemorrhage is not uncommon. Perforation produces the usual symptoms. The resulting fistulation will be discussed under fistulas. Symptoms of obstruction due to this ulcer do not vary from those due to

or as a result of infection. A history of allergic reactions elsewhere can usually be obtained. Pruritus ani is a particularly troublesome allergic manifestation. With or without colonic involvement the symptoms closely resemble those of ulcerative colitis. As in other diseases associated with diarrhea and pain there is usually a more or less definite psychic impact accounting for the popular feeling that the disease is of psychosomatic origin. The complications will each produce their special symptoms such as those of appendicitis or of perforation or obstruction when due to other causes. Fistulas to other parts of the intestine will occasion more severe symptoms. fistulas to the bladder, pelvis or skin will produce symptoms there. Evidence of the other complications mentioned above should be watched for.

### Physical Examination

There may be no abdominal findings or there may be tenderness and sometimes a palpable mass over the area of involvement. With obstruction distention may be prominent with perforation or appendiceal involvement signs of peritonitis may be present. General emaciation and pallor are usually marked as are evidences of nutritional deficiencies and other allergic manifestations.

*Proctoscopy* usually shows normal findings although the diarrhea may cause irritative changes, redness and edema and the surrounding skin changes of pruritus.

*Intubation* is used only when obstruction is suspected and suction must be used with great caution to prevent perforation.

### Laboratory Examinations

*Stool examinations* will show much mucus, some cellular exudate and occult blood with visible blood only in colonic involvement or in the ulcerative form of enteritis. Fat granules and in severe cases also starch granules and meat fibers may be found.

*Stool cultures* show no constant bacterial content except in secondary infection when streptococci, staphylococci, Welch's bacillus or other organisms may be found.

The *urine* will show concentration and in severe cases evidences of nephrosis or actual nephritis.

*Blood Examination.* Anemia becomes progressive only moderately hypochromic because of the marked dehydration. Leukocytosis is usually present. The sedimentation rate is increased. In severe cases hypoproteinemia, hypoprothrombinemia and hypoglycemia will develop.

### X-ray Examination

It is always well to do a barium enema first to avoid impaction if obstruction should be present. This study will reveal whether there are

changes and cicatrization Spread does not occur throughout the small intestine as there are skip areas with no involvement followed by areas of marked disease The findings suggest those of collagen diseases elsewhere Long spontaneous remissions are not uncommon During the intervals between attacks there may be no gross evidence of disease although careful study will show some fibrotic and polypoid changes Malignant degeneration practically never occurs

### Complications

*Local Complications* Ulceration may develop to the point of perforation more frequently walled off resulting in further fibrosis and cicatricial contraction with partial obstruction Secondary infection causes localized abscesses which may rupture into neighboring organs or the skin accounting for the fistulas which are a feature of the late stages Appendicitis is a frequent complication Fistulas are apt to develop after appendectomy or removal of an involved segment Ischiorectal abscess and fistula is a serious complication Such other conditions due to collagen disease as rheumatoid arthritis periarteritis nodosa and skin changes are prone to occur Ulcerative colitis may also be present Malignant changes have not been found

*General Complications* In careful autopsy studies by Barger et al it was found that about half of the deaths were due to peritonitis Changes in other organs probably attributable to allergy or severe malnutrition include fatty infiltration and focal necrosis in the liver pancreatic fibrosis renal inflammation and tubular degeneration vascular thromboses and amyloidosis

### Etiology

Much confusion exists over the cause of this disease Some agent in the blood or in the intestinal content has been blamed and infection and infestation have been suggested Allergy has not been ruled out and the conception that the disease is an allergic phenomenon fits in with its clinical behavior as well as its pathological findings (see under Allergy p 81)

### Symptoms

At the onset food poisoning or virus infection is usually suspected In mild cases there may be no symptoms evidence of the disease being discovered upon routine x-ray study The usual symptoms consist of diarrhea up to ten or twenty stools per day with colicky midabdominal or right lower quadrant pains and marked loss of weight and strength with evidences of nutritional deficiencies Rarely is there blood in the liquid stools In children there is a marked retardation of growth and development Fever and chills may occur as part of the allergic reaction

there is involvement of the ileum. Careful x ray study will decide the diagnosis. The findings on proctoscopy are also significant.

*Tuberculosis* in the past was a difficult condition to differentiate and many cases were treated as such. Today, however, it is a rare condition although it should be borne in mind. It is usually secondary to pulmonary tuberculosis.

*Ileal necrosis* and cicatrization following partial mesenteric infarction may be confusing. A phlegmonous inflammation of the ileocecal region with ascites is a complication in severe cases of infectious hepatitis and is usually terminal.

*Cancer* is an important condition to rule out. Although in general ileitis occurs mostly in young adults or children, cancer is occurring more in this age group. The symptoms may be similar but careful x ray study and proctoscopy should clear up the diagnosis.

*Lymphogranuloma venereum* occurs mostly in the distal half of the colon, especially the rectum but never in the small intestine.

### Treatment

*Surgical Treatment* When this disease was first described, resection of the affected gut was held to be the only treatment. Because of poor results with frequent recurrences at the stoma and elsewhere, the development of fistulas and the serious interference with the patient's nutrition when extensive resections had been done, surgery has been relegated to the treatment of complications. These include obstruction, perforation or fistulation and anorectal abscesses and fistulas. The simplest operation to promote drainage is often preferable to attempts at resection. An ileostomy well above the topmost lesion seems simple but is of course a fistula in itself and will not prevent other fistulas or other areas of involvement from occurring. Anastomosis between the small intestine above the lesion and the transverse colon is now more frequently done. Simple drainage for perforation or abscesses may be necessary primarily. In the past patients were sometimes subjected to multiple operations before death.

*Medical Therapy* **ALLERGIC TREATMENT** The best although not wholly satisfactory results are obtained by treating the patients primarily as allergic cases. A careful allergic study by elimination and addition diets and avoidance of foods shown to be causing symptoms is important. Areas from which the patient may be absorbing proteins to which he is sensitized such as so called focal infections, teeth, nose, throat, sinuses, pelvis, rectum or chest must be removed or actively treated. The subject of treatment for allergy is fully discussed in the chapter on this subject already referred to above.

**GENERAL CARE** The patient's general dehydration, malnutrition, toxemia and psychic disturbances must be carefully and individually treated.



Figure 62 Regional enteritis 1 String sign 2 3 4 other narrow areas separated by skip areas b Note areas of fragmentation in other areas (deficiency pattern)

areas of involvement in the colon as shown by narrowing ulceration or fistulation. Regurgitation of the barium into the ileum will often show the string sign in terminal ileitis which is due to the marked narrowing of the involved portion of the small intestine.

Study after an opaque meal should always follow the enema study. Fluoroscopy and films at frequent intervals in different positions will disclose dilatation behind narrow areas, irregular narrowings down to string size in the affected areas and skip areas of normal appearing intestine except that the so called deficiency pattern or irritability and fragmentation is of frequent occurrence in the entire small intestine (Fig. 62). This makes it necessary to use extreme care to avoid making a diagnosis of sprue. Fistulas can often be clearly demonstrated although they are sometimes overlooked. In suspected obstruction or perforation only scout films should be taken. After resections have been performed recognition of recurrences is extremely difficult.

### Differential Diagnosis

*Appendicitis* is most frequently the first diagnosis and many patients probably more than one third have appendectomies performed. Even at operation the diagnosis is not always made. X-rays even scout films may obviate a useless operation although appendectomy may be necessary because of complicating acute appendicitis.

*Ulcerative colitis* has symptoms which are liable to be similar especially in the early stages. Also in about 20 per cent of ulcerative colitis

careful study of the pathology and etiology is necessary for successful management

### Pathology

The term "ulcerative colitis" does not embrace a pathological entity. Ulcers due to specific organisms with surrounding or generalized colitis may occur in any part of the colon. The dysenteries are really colitides due to bacteria of the *Shigella* group or to *Entamoeba histolytica*. Acute infections with *Salmonella* organisms, *Leishmania donovani* and with *Bacillus pyocyaneus* produce an ulcerative colitis which may become chronic. Lymphopithia venereum, syphilis, tuberculosis and direct infection of the bowel wall with pyogenic or mycotic organisms or even typhoid organisms may also produce what in the general sense may be called ulcerative colitis. Even the presence of an ulcerating carcinoma will produce a surrounding colitis and it is sad to relate how frequently patients with carcinoma are treated as ulcerative colitis cases until beyond the stage of operability. As a rule today when speaking of ulcerative colitis we mean a disease which runs a definite course but for which no specific organism has been found responsible and whose cause is in dispute—hence the term "idiopathic ulcerative colitis." Kantor suggested the term "colitis gravis" as a better name.

The pathologic lesions of such an ulcerative colitis occur in stages and may at first be found only in one segment of the bowel, often the rectum and sigmoid, and may remain localized or spread later to the entire colon and even to the terminal ileum. It has been suggested that ulcerative colitis may be classed among the collagen diseases.

In the early stages hyperemia, hypersecretion and mucosal edema are present and a punctate rash resembling herpes is usually seen. As a result of localized ischemia areas of necrosis develop and slough out leaving ulcerated areas which when they coalesce cause extensive denudation of mucosa with small islands or tabs of mucosa still visible. These denuded areas resemble eczema in the skin. They may bleed on slightest trauma or the sloughing out of the walls of small blood vessels may produce more profuse bleeding. Deep sloughing, often causes destruction of interstitial tissues resulting in the breaking or obliteration of the basement membrane. This may lead to single or multiple perforations with consequent local or general peritonitis and later adhesions, deformities, kinks or obstructions. Rarely fistulas may result extending into the pelvis, the perineal region or ureter and even producing subphrenic abscess. Induration with infiltration by leukocytes, often demonstrable as eosinophils, may disappear with healing or may result in fibrosis with narrowing or stiffening and shortening of the colon. The mucosal lesions may disappear entirely or with the long continued irritation islands of mucosal remnants may undergo polypoid change. It



A *well balanced diet* with avoidance of allergens is most important and the chapter on allergy gives details of this diet. Added *vitamins and minerals* are necessary also guided by consideration of allergies. At the onset *parenteral feedings* of glucose and electrolytes may be necessary. The use of protein dialysates must be guided by knowledge of the patient's allergies since a preparation made from milk may cause serious symptoms in a patient with milk allergy. *Blood transfusions* while valuable are subject to the danger of allergic reactions to food in the donor's diet as well as the danger from transmission of hepatitis which in a debilitated patient might result fatally. The use of *sulfonamides and antibiotics* except when secondary infection has been definitely demonstrated is also not desirable from the allergic standpoint. They may cause symptoms as serious as the disease itself. X-ray therapy cautiously used may reduce cellular infiltration and edema temporarily but can do harm. *Corticosteroid* therapy as in other allergic diseases though often showing *marked improvement at first* does not prevent spread of the disease. *Antihistaminic drugs* rarely relieve symptoms. Medication for the diarrhea is markedly unsatisfactory. Various soothing medications demulcents antispasmodics anticholinergics and narcotics have been tried. *The elimination of the allergenic cause is usually most successful* in slowing down the frequency of defecation and irritation from feces.

### Prognosis

*This is a serious disease and although at times it may clear up spontaneously or after such treatment as outlined above the tendency to recurrences is the same as in other allergic diseases.* However with more or less constant watching for the development of new food allergens and the elimination of focal infections as they occur a good prognosis can be made.

### ULCERATIVE COLITIS

For the past twenty years or more an extensive literature has been accumulated on the subject of this rare disease. The average general practitioner may not see a case of ulcerative colitis more often than once in a year or longer. It is important that such a case be recognized and properly evaluated so that adequate treatment may be instituted before serious complications develop. It is also important to realize that the bloody diarrhea which is the characteristic symptom in nearly all cases also occurs in cancer and that cancer must be ruled out in every case. The varying opinions about the etiology and treatment of this disease are reflected in the literature on the subject. The result is that many radically different kinds of treatment are carried out. Patients travel from clinic to clinic and city to city in search of a cure and frequently wind up with multiple operations leaving them crippled for life. A

The only factor which I have found to be almost invariably the cause of this disease has been *allergy* usually to foods although occasionally to pollens dusts molds fungi bacteria or their products as in focal infections. Endocrine or hormonal secretions would seem also to be the allergens to which the patient may become sensitized. The proof of the role played by the specific substances is not a simple matter. It requires patience and thoroughness on the part of physician and patient to establish the cause or causes but once these have been determined their effect on the bowel is convincing as is seen by roentgen study and proctoscopy. Theoretically comparison of the conditions in the intestine with those in another well known shock organ the respiratory tract helps to explain a number of the findings in ulcerative colitis. In asthma for instance severe specific acute infections often precede the onset of the original allergic symptoms secondary infections may result in masking of the cause removal of focal infections will occasionally result in cure neuropsychiatric symptoms may abound and when instead of occurring periodically the symptoms become constant and severe structural changes usually follow.

### Symptoms

The history is important. A family history or previous personal history of some form of allergy is suggestive. The onset of the disease may occur in several ways.

1. It may be preceded by a more or less long continued history of attacks of diarrhea or other abdominal symptoms often attributed to specific dietary indiscretions the patient stating that milk or coffee or some other food acted as a laxative or cathartic.

2. There may be a gradual onset with mild diarrhea in the early stages gradually becoming more severe with bleeding increasingly noticeable.

3. The symptoms may occur sooner or later after an acute dysentery an acute gastroenteritis or food poisoning.

4. There may be a definite seasonal occurrence of the attacks suggesting allergy to a certain food or foods taken by the patient either directly or in the milk of cows when these food ingredients are in season. It may be due to pollen or other inhalants.

The symptoms are readily explainable on the basis of the pathologic findings. The allergic reaction in the mucosa causes spasms and hypermotility which produce pains tenesmus and diarrhea the number of loose or watery stools varying from three or four to twenty or thirty per day. The sloughing and exfoliation with bleeding and excessive production of mucus add blood and mucus to the stools and eventually result in frequent bloody rectal discharges independent of fecal admixture. The patient may actually have only one real stool in twenty four or

has been suggested that cancer may develop in these pseudopolyps but it is much more probable that in the rare cases in which carcinoma follows ulcerative colitis real pre existing adenomatous polyps are the cause. Polyps are not uncommon in the normal colon. More or less bacterial invasion may be found in the tissues and lymphadenitis may be demonstrable in the mesenteric glands. The early changes are identical both grossly and microscopically with the changes described by Gray, Walzer, Harten and their associates. They reported on their experimental work in gastrointestinal allergy in which they used the mucosa over human hemorrhoids and colostomies and the intestinal walls of the Rhesus monkeys sensitized by passive transfer to observe the pathologic changes caused by ingestion of the food to which the tissues had been sensitized. These changes were produced by a single swallow of the antigen. It can easily be imagined what the effect of such insults frequently repeated would be in producing the more extensive changes in severe cases.

Not only all the active lesions can be caused by allergic reactions but scarring, fibrosis and narrowing can be produced without secondary infection. Similar changes in allergic conditions in the lungs and eye may be cited as examples.

### Etiology

There is a surprising difference of opinion as to the cause of ulcerative colitis. Because except in the cases of specific organisms mentioned before the colon bacillus is usually the only or the predominant organism found it was assumed for a time that in some persons it could become pathogenic. The enterococci, normal inhabitants of the ileum and usually not viable in the colon except when carried through it rapidly as a result of diarrhea, were next implicated and one of this group was considered specific. Various other organisms, viruses and fungi have been suggested at different times. The fact that the disease occurred in many patients who had previously had dysentery implicated the dysentery organisms. The food and vitamin deficiencies, actually the result of a lack of absorption, impelled some clinicians to include ulcerative colitis among the deficiency diseases. The usual neuropsychiatric manifestations which should be expected to occur in patients with such severe gastrointestinal symptoms have been interpreted by the psychiatrists as the causative factors and in some cases specific changes in colonic innervation have been postulated. Psychic traumas at times may act as a trigger to start an attack or exacerbation. The often dramatic relief of all symptoms following eradication of focal infections has resulted in efforts at isolating a specific organism in the foci but without result. The resemblance to collagen diseases has been mentioned. Allergy has been suggested as a cause of collagen disease.

looks like raw meat coated with blood mucus and sloughing mucosa showing yellowish discharges of pus only where secondary infection has occurred. Rarely such findings are present without diarrhea. These findings are in contrast with the discrete ulcers of amebic dysentery scrapings from which will show motile amebae with enclosed blood cells and the multiple small or coalescent ulcers with purulent and bloody exudate seen in bacillary dysentery. Tuberculous and syphilitic ulcers and the lesions of lymphopathia venereum which may be somewhat confusing because of their variability are usually accompanied by other general manifestations of these diseases and appropriate tests will establish the diagnosis. Rectal and sigmoid neoplasms both cancer and multiple polyps which often produce symptoms similar to those of ulcerative colitis must be ruled out.

Through the proctoscope biopsy specimens may be removed. Smears should be prepared from scrapings particularly to search for carcinoma cells or for the eosinophils frequently seen in ulcerative colitis. Material for cultures and further cytologic studies should be obtained by means of long pipets, syringes or sterile swabs protected from contamination in long sterile tubes. Repeated proctoscopies are a guide to improvement under treatment and to the effects of feeding of suspected allergens.

### X-ray Examination

An opaque enema study should not be the full extent of the examination unless it has shown a definite obstruction of the colon. A careful gastrointestinal series with films taken at frequent intervals after the barium meal will show the often concomitant lesions of the small intestines or the effects of food and vitamin deficiencies. The highly irritable colon may empty in twenty-four hours but at times the induration, adhesions, pseudopolyposis or other changes may cause delay of the barium meal in the colon even though the patient is having frequent bloody discharges from the rectum. Neoplasms both benign and malignant must be ruled out.

The roentgen findings are characteristic (Fig. 63). At first colonic irritability and spasms occur followed by disappearance of haustrations when the edema and induration cause thickening of the bowel wall. The bloody mucus coated mucosa produces the fuzzy outline seen in the films later sloughs produce a ragged outline. Progressive developments lead to a general shortening of the colon, a gradual decrease in the size of the lumen and evidences of kinks, adhesions, walled off perforations or obstructions. Repeated roentgenographic studies during the course of the disease should be used to register the improvement which is to be expected under adequate treatment.

If the patient's condition permits a cholecystographic study should

forty eight hours while thirty to forty of the bloody rectal discharges are taking place each day

Anorexia disgust for food and the nervous symptoms including a depressive state resulting in insufficient food intake the failure of digestive secretions and the rapid progress of food over the diseased intestinal mucosa preventing adequate absorption—all combine to cause emaciation dehydration and evidences of food and vitamin deficiencies Absorption of toxic material from denuded areas and secondary infection will produce fever adenopathy and toxic symptoms Skin complications such as eczema erythema nodosum and pyoderma gangrenosum may occur The neuropsychiatric symptoms which may at first interfere with care of the patient often change for the better with unbelievable rapidity as the patient's general condition improves under proper care Mesenteric lymphadenitis produces symptoms which may suggest an acute condition of the abdomen and perforation though rare may be rapidly fatal Extreme distention may precede perforation I saw a patient with ulcerative colitis who died after perforation of an old sigmoid diverticulum

### Physical Examination

*Abdominal examination* may disclose nothing abnormal may show distention localized or general tenderness or rigidity and at times the thickened or spastic colon may be palpable and tender *General examination* will usually reveal the presence of focal infections evidences of allergic reactions in the skin or other parts of the body and in severe cases will disclose the emaciation dehydration and evidences of food vitamin and mineral deficiencies already mentioned It is important to ascertain at the start whether the patient has any organic lesions in any part of the body so that complications may be guarded against and treated early if they develop Search for focal infections in the mouth nose and throat pelvis and genitourinary tract should be thoroughly carried out

*Rectal Digital Examination* This may show sphincter spasm which may later change to marked relaxation producing incontinence Hemorrhoids usually develop and may become so large as to require treatment Cryptitis occasionally associated with greatly enlarged papillae which may protrude is also a distressing complication Rectal abscesses and fistulas are frequently found The digital examination may also disclose strictures polyps and pelvic tumors and in many cases previously and erroneously treated for ulcerative colitis we find a hard irregular carcinomatous infiltration

*Proctoscopic Examination* The findings in typical ulcerative colitis are those which were described in discussing the pathology of this disease In severe cases there will be seen only a bleeding surface that

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a

b

**Figure 63** Ulcerative colitis—barium enema *a* Early colitis. Note general atony, few shallow haustrations, fuzzy outline, few rectal ulcers (at arrows). *b* Late advanced case. Note definite shortening, absence of haustrations, narrow lumen, pseudopolyps (at arrows).

also be made. All patients should have chest films to rule out tuberculosis or any conditions which might be complicating the intestinal condition.

### Laboratory Studies

These data are of considerable help. Blood cell counts, not only to determine the degree of anemia, which is often extreme, but also to show the eosinophilia so often present, are important. Chemical studies of the blood have shown no constant findings, but may show acidosis, diminished chlorides, calcium and phosphorus, renal insufficiencies, and varying degrees of vitamin deficiency. Prothrombin determinations and other coagulation tests usually show normal findings. The sedimentation rate is nearly always increased. Serologic tests to rule out syphilis, lymphopathia venereum, leishmaniasis, and perhaps amebiasis, agglutination tests for dysentery, typhoid, and *Salmonella* groups, and the Frei tuberculin and mycotic skin tests should be performed routinely. Hyperglycemia is occasionally found in severe cases and is usually due to liver damage.

**Fractional Gastric Analysis.** By using histamine as a stimulant to gastric secretion, variations in acidity may be found. Absence of an achlorhydria does not necessarily rule out the possibility of absorption of unchanged protein, as shown by Walzer and Gray. Mere so-called gastrogenous diarrhea never accounts for the severe symptoms of ulcerative colitis, and this is also true of pancreatic deficiencies.

*Stool Examinations* Such studies are important and should be performed daily for at least the first ten days. Blood, mucus and mucosal shreds with or without admixed feces are usually found. Cysts, ova or parasites and yeasts should be looked for and undigested food particles should be noted. Although bacteriologic studies are best carried out primarily on specimens removed through the proctoscope as previously mentioned they may be continued on specimens of stools thus avoiding repeated instrumentation. In the cases of specific bacterial colitis the effect of treatment on the flora may be checked, bacteriophages may be looked for and vaccines may be prepared. The improvement in the general appearance of the stools, the disappearance of blood and the gradual increase in consistency in cases under proper treatment are most gratifying. For a while much attention was focused on lysozyme determinations but the most that can be said about them is that notable increases in the concentration of this mucolytic enzyme seem at times to be associated with intestinal ulceration.

#### Allergy Study and Treatment

This may be started during the period of other examinations. Authorities agree that skin and passive transfer tests are of no value in determining food sensitivity but may be useful if the patient is also sensitive to pollens, molds, fungi, dust or emanations. Bacterial allergy is difficult to demonstrate but removal of infected teeth or tonsils may result in prompt disappearance of the ulcerative colitis thus suggesting sensitivity to bacteria, their products or the products of dead tissues produced by the infection. Referral to an allergy department or specialist does not answer the requirements. In a condition as severe as ulcerative colitis the food causing the severe reaction must be one which is being taken daily by the patient. In my experience milk has been found to be one of the offending foods in over 50 per cent of cases and the only one in nearly 40 per cent. Other sources are wheat in 18 per cent, tomatoes in 15 per cent, oranges and potatoes in 12 per cent each and eggs in 9 per cent. During the general study of the case I usually eliminate milk and milk products from the diet which should be well balanced and contain an adequate amount of residue. In milk sensitive patients there will be considerable improvement in symptoms by the time the study has been completed and thereafter a careful record of all foods eaten and of the frequency and character of bowel discharges and general symptoms will usually disclose other allergens.

When no improvement occurs under the milk free diet a more detailed study is indicated. Instead of slow and often inadequate elimination diets we have been using a diet which contains none of the foods usually eaten daily and which have been shown to be a frequent cause of ulcerative colitis. In other words we eliminate altogether milk, eggs,



wheat potatoes oranges and tomatoes and their products and in order not to have too many offending foods to deal with we give a diet consisting of only about a half dozen foods which after careful inquiry we find the patient has not been in the habit of taking regularly and which he has not ever suspected of causing any symptoms Since such a diet is necessarily poorly balanced is often not sufficiently nourishing and may cause some loss of weight *it is used for only a few days* Careful individual study by the physician intern nurse or other daily attendant is absolutely necessary A sample diet of this kind is allergy test diet number 2 (see p 93) consisting of five foods gelatin rice rye peas and raspberry (or other vegetable or fruit) given as follows

### Allergy Test Diet

<i>Breakfast</i>	Boiled rice Ry Krisp gelatin drink raspberries
<i>Lunch</i>	Peas Ry Krisp gelatin drink raspberries
<i>Supper</i>	Same as lunch or breakfast
<i>Between meals and at bedtime</i>	Gelatin drink and Ry Krisp

The gelatin drink is made by adding 1 teaspoonful of powdered gelatin to a fruit juice made of the fruit or berry used in the diet sweetened and fortified with dextrose Sixty to 80 gm of gelatin and 100 to 150 gm of dextrose can be used in this diet to supply protein and to bolster the caloric intake

If this diet contains no food to which the patient's colon is sensitized definite improvement can be seen within two or three days Stools become a little less frequent and bloody the patient becomes brighter and says he feels better in general and fever may show a downward course If this improvement does not occur it means that the patient is allergic to one of the five foods being taken that he is allergic to some other factor (bacteria pollens or the like) or that the case is not one of allergic origin The restricted allergy diet must not be kept up too long in such cases although at times it may require five or six days to get definite improvement

When improvement occurs new foods are added daily with careful observation for even a slight exacerbation as a result of any particular addition By repeated trial allergens can be conclusively demonstrated In view of the fact that the six foods previously mentioned are usual offenders they should not be tried out until the patient has achieved a maintenance diet by the addition of meats cereals vegetables and fruits in considerable variety A complete study for food allergy may take from two to four weeks Finally the patient is placed on a well balanced diet with caloric values according to his requirements and it is surprising to see the improvement achieved on a diet free of offending allergens even though the diet contains considerable quantities of so called roughage

### Treatment

Although elimination of the offending foods from the diet is essential and removal of focal infections may be of the greatest value in treating a patient with ulcerative colitis there are definite indications for other therapeutic measures

*Desensitization* to offending foods by injection of food proteins or by feeding increasing quantities has not been found of value. However when allergy to pollens, molds, dusts or other inhalants is demonstrated to be the cause of the colitis, desensitization may be of great value (See chapter on Allergy 94)

*General Treatment* Rest, physical and mental, is indicated. Fluids to replace those lost by the diarrhea are necessary and at first must be given parenterally. Transfusions, plasma and glucose and saline solutions with added ascorbic acid should be used to reestablish water and electrolyte balance and overcome the anemia. Protein hydrolysates, amino acids and albumin are of help, but in milk sensitive patients the milk derivatives may cause untoward reactions and may aggravate the bowel lesions. This may be true of blood from a donor who has eaten food to which the patient is sensitized. Hepatic damage is usually repaired gradually on a balanced diet. Vitamins, minerals and endocrine and hormone products must be given at first parenterally. Hygienic care, fresh air, sunshine, ultraviolet radiation and oral hygiene are of great value.

*Symptomatic Treatment* In order not to disguise the effects of the allergic study this should be kept at a minimum but in the early stages some sedatives and antispasmodics may be prescribed. Coagulants may be indicated. Skin and rectal conditions may require attention. Rectal instillations of 4 to 6 ounces of 10 per cent gelatin solution at temperatures of 110 to 115° F. to be retained may help to control bleeding and allay irritations (see p. 47).

*Immunologic Treatment* The use of vaccines, serums, bacteriophages or bacterial filtrates is of value in some cases. *Artificial pyrexia*, popular twenty five years ago, is still being used and is useful in any allergic state. In the acute, fulminating case with marked toxemia and skin infection, arthritic or other symptoms, *steroid therapy* may dramatically relieve the symptoms. It should not be continued for more than a few days, however, because perforation has occurred from its use.

*Specific Therapy* This is of course necessary for amebiasis and a course of amebicidal therapy is recommended by some as good routine procedure to rule out the possibility of unrecognized amebiasis. Some authorities also recommend routine treatment with sulfonamides which are necessarily of value only when the colitis is specifically bacterial in origin or when there is a complicating secondary infection. In such cases antibiotics have also had their advocates but the diarrhea produced

wheat potatoes oranges and tomatoes and their products and in order not to have too many offending foods to deal with we give a diet consisting of only about a half dozen foods which after careful inquiry we find the patient has not been in the habit of taking regularly and which he has not ever suspected of causing any symptoms. Since such a diet is necessarily poorly balanced is often not sufficiently nourishing and may cause some loss of weight it is used for only a few days. Careful individual study by the physician intern nurse or other daily attendant is absolutely necessary. A sample diet of this kind is allergy test diet number 2 (see p 93) consisting of five foods gelatin rice rye peas and raspberry (or other vegetable or fruit) given as follows

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occurred (4) A new bacterial mycotic or parasitic infection has been acquired

In an intelligent patient under careful although not necessarily close supervision by a clinician familiar with allergic problems the prognosis in ulcerative colitis can be said to be decidedly favorable not only for restoration of a well functioning colon but also with respect to the danger of severe exacerbations

#### ACUTE PSEUDOMEMBRANOUS ENTEROCOLITIS

This disease is one of the most serious complications following antibiotic therapy especially of the broad spectrum type although at times penicillin and streptomycin have been the cause Though it may occur after such medication given for any cause it seems to be most frequent following operations during and after which the antibiotics were used for prophylaxis against infection Ordinarily the diarrheas following antibiotic therapy can be attributed either to allergy or to alteration of the intestinal flora The *Proteus* and *Pseudomonas* organisms are usually found Such diarrheas usually clear up spontaneously although they may continue for six or eight weeks or more The pseudomembranous variety has been found to be caused nearly always by the *Micrococcus pyogenes* (*Staphylococcus aureus*) found either alone or associated with gram negative bacilli The bacteria may also be found in the blood The reason for the sudden severe infection is that these organisms have become resistant to the action of the antibiotic and begin to multiply and invade as soon as other susceptible bacteria have been destroyed The incubation period is from a few hours to a month or more usually from three to seven days

#### Pathology

The entire small and large intestines and even the stomach and esophagus may be involved or there may be involvement of only varying lengths of bowel Progressively there is congestion ulceration denudation and sloughing of the mucosa frequently with gangrenous areas even without obstruction A fibrinous membrane may be adherent to the mucosa and when peeled off will show pure pus underneath With bacteremia infections of the kidneys lungs and spleen are common complications

#### Symptoms

The onset is usually acute and severe rarely insidious and death frequently occurs within a few hours to a week or ten days Fever nausea vomiting colicky pain and diarrhea occur early and the patient may go into shock from the violence of the onslaught The stools may resemble the rice water stools of cholera and may contain free pus and pieces of fibrinous membrane Rapid dehydration occurs with disturbance of water and electrolyte balance oliguria uremia and death

by the broad spectrum antibiotics themselves may cause serious complications. Some of the newer antibiotics do not have this effect. Whether or not the bowel content can be sterilized is of comparatively little moment in a case of allergic colitis except with secondary infection. In such infection in the rectum instillation of saline suspensions of antibiotics is useful. There is always the danger of the patients being or becoming allergic to any drug.

*Psychotherapy* Popular today in a great variety of chronic illnesses psychotherapy is also being strongly advocated for this disease. Without doubt the extremely ill patient with ulcerative colitis requires proper psychologic supportive care but elaborate psychologic studies often make the patient more miserable. It is remarkable how rapidly the patient's mental attitude improves when symptoms begin to subside.

*Surgical Treatment* In uncomplicated cases there is no excuse for operation. Some surgeons recommend operation even colectomy in the acute fulminating toxic case but it is a risky procedure. Even such simple operations as hemorrhoidectomy and fistulectomy may cause a fatality in the fulminating case. In such cases forcible sphincter dilatation (under anesthesia) affords relief from spasm and may promote resolution. If as a result of prolonged or repeated attacks the colon has become obstructed or irreversibly crippled as a result of scarring, fibrosis, adhesions or extensive polypoid changes operation must be considered. *Acute perforation unless promptly walled off is also an indication for operation.*

Today the operation of choice is a total colectomy with of course a permanent ileostomy. Temporary ileostomy or colostomy to put the colon at rest was formerly popular but was not successful and has been abandoned. Surgeons often speak of the joy of living with an ileostomy. Occasionally after operation cancer occurs or the ileum becomes involved and the patient is worse off.

### Prognosis

Mortality in our cases treated as outlined has been under 5 per cent and is then due to complications. When the treatment has been faithfully carried out and the patient has been placed on a regimen based on the facts learned during the study and early treatment there may be no exacerbations or symptoms may recur as they do in asthma, hay fever and other allergic conditions. If at the onset of an exacerbation a careful search for the cause is instituted it will usually be found that (1) The patient deliberately or inadvertently ate one of the foods to which he had been found to be sensitized. (2) Some new sensitivity or one not previously recognized such as pollen sensitization may have developed. Antibiotics given for an acute upper respiratory tract infection are frequently the cause. (3) Activity at some focus of infection, an abscessed tooth, an acute upper respiratory tract or acute pelvic infection has

but rarely sufficient to result in exsanguination. Abdominal pain is often the first symptom and may be generalized or localized in the epigastrium or midabdomen. It may be so severe as to suggest an abdominal calamity. Abdominal examination may show only moderate distention; tenderness may not be localized and rigidity is unusual. Usually there is a neutrophilic leukocytosis as high as 25,000 cells per cubic millimeter and proteinuria is a common finding.

### X-ray Examination

Scout films may show only a moderate distention and rarely fluid levels may be seen. The patient is usually too weak to permit further x-ray study.

### Treatment

The treatment is supportive following the same principles as described for the pseudomembranous variety. Nearly all patients die within one to five days. Rarely has a patient died in a second attack.

## BACILLARY DYSENTERY

Although generally considered a tropical disease and often named after various tropical countries, bacillary dysentery may occur sporadically in various parts of the world with occasional epidemics especially among school children. It is usually due to poor sanitation as in armies, institutions and communities in which prophylaxis is neglected. It is caused by the *Shigella dysenteriae*, many strains of which are known. They are divided into three main groups: Shiga's bacillus causing epidemics in the tropical Far East; Flexner's bacillus seen in northern climates mostly in the Western Hemisphere; and the Sonne dysenteriae endemic everywhere.

### Pathology

These bacilli produce both endotoxins and exotoxins which cause a toxemia in addition to attacking the colon and at times the terminal ileum. After an incubation period of hours to a week they attack first the mucosa and cause more or less severe inflammation, suppurative exudate, hemorrhagic areas and necrosis. There may be produced an organized coating or membrane with ulcers beneath and a tendency to bleed. Coalescing ulcers may result in sloughing, cicatricial contraction and retention cysts. The lymph nodes become involved, the spleen may become enlarged and there is congestion of the kidney, liver and adrenals. In severe cases there may be a bacteremia. In some cases chronic relapsing diarrheas may occur with the findings of ulcerative colitis. Felsen considered the *Shigella* to be the cause of so-called nonspecific ulcerative

**Diagnosis**

The patient may die before the diagnosis is made especially in cases with an *insidious onset resembling a virus infection* occasionally with not even a diarrhea at the onset. The abdomen may show only distention and moderate tenderness or may be severely tender and spastic almost suggesting an abdominal calamity. The patient is usually too ill for an x ray study beyond scout films which may be negative. Chest x rays may show multiple abscesses the urine will disclose renal infection. *Stool and blood cultures* will frequently reveal the *M. pyogenes* (*Staphylococcus aureus*) usually in pure culture although occasionally combined with *Pseudomonas pyocyanea*. The organisms should immediately be tested *in vitro* for sensitivity to antibiotics.

**Treatment**

There is no time to lose so that treatment must be instituted even before cultures confirm the diagnosis. Erythromycin in full doses will usually do no harm and may be lifesaving. General measures to correct the effects of dehydration as mentioned above must be started at once. High caloric well balanced liquid feedings by mouth as recommended in postoperative cases (see p. 276) and additional parenteral feedings and transfusions may be helpful. Stimulation such as Levophed 4 cc of the solution added to 1000 cc of 5 per cent dextrose in water or saline solution given slowly by vein may be necessary for shock. Renal and pulmonary complications require attention.

**Prognosis**

In spite of all the measures recommended above most cases end fatally.

**ACUTE HEMORRHAGIC ENTEROCOLITIS**

This disease resembles acute pseudomembranous colitis but no definite etiologic factor has been found. It occurs mostly in middle or old age in patients with *chronic cardiovascular renal or other diseases*. At times it follows antibiotic therapy. In the absence of specific organisms causing the disease such reaction must be considered allergic. The essential pathological finding is a general breakdown of interstitial capillaries resulting in necrosis sloughing and hemorrhage. This capillary breakdown has been ascribed to senility to malnutrition congestion and anoxia. Occasionally there is bleeding elsewhere with ecchymoses in the skin or a subarachnoid hemorrhage.

**Symptoms**

The onset of symptoms is abrupt often accompanied by shock and a subnormal temperature. Diarrhea is usually present often bloody.

**Treatment**

*Prophylaxis* is accomplishing wonders today. With improvement in sanitation, disinfection of feces, elimination of carriers and flies, and care in food sterilization, much has been accomplished. Preventive inoculation was shown to be of great value during the war. All persons going to endemic areas are now required to be immunized and while in the areas many take antibiotics as an additional precaution. Diarrheas developing in tourists returning north may be the result of the antibiotics rather than of dysentery.

*General Treatment* Of great value are parenteral fluids, transfusions, glucose, protein dialysates, and electrolytes, followed by an adequate, well-balanced diet, preferably soothing, similar to an ulcer diet (p. 267), with added vitamins and minerals. Bismuth, kaolin, and pectin help to soothe the irritated mucosa. Rectal bleeding can be at least partly controlled by warm retention enemas of 10 per cent gelatin solution (see p. 47).

*Specific Treatment* Formerly antidyenteric serum was much used and reduced mortality in severe cases. Bacteriophage, when obtainable, is of great value, almost magic in its effect. Full doses of broad spectrum antibiotics, selected after tests *in vitro* for specific effect on the offending organism, have helped to reduce the mortality.

*Complications* may require surgical care. Subsequent ulcerative colitis must be treated in the usual manner.

**Prognosis**

Today, with the broad spectrum antibiotics available, bacillary dysentery is not as formidable a disease as formerly. With adequate prophylaxis it may eventually be stamped out, as typhoid has been.

**INTESTINAL ALLERGY**

Both the large and small intestines are frequently the site of allergic reactions. The whole subject is fully discussed under Gastrointestinal Allergy (p. 87) and under Ulcerative Colitis (p. 352) and Regional Enteritis (p. 347), which are severe allergic manifestations.

**COLLAGEN DISEASE**

The colon and appendix and more rarely the small intestine have been involved in diseases recently classified as collagen diseases. Lesions in the intestines have been described which resemble scleroderma, dermatomyositis, and polyarteritis nodosum, and are usually found in conjunction with these collagen diseases elsewhere. It has also been suggested that regional enteritis and ulcerative colitis may be classed as collagen diseases. The whole conception of collagen disease is still rather vague (see p. 99).



colitis although he was never able to demonstrate the organisms to prove this theory. Amebic dysentery must not be confused with bacillary dysentery. It is discussed in the chapter on intestinal parasites (p. 139).

### Epidemiology

The organisms are transmitted through stools so that infection occurs from the drinking of water contaminated by feces or from milk or other foods taken unheated after being contaminated by food handlers or flies.

### Symptoms

The disease occurs in varying degrees of severity. Mild cases occurring in the North and also occasionally in Northerners visiting the tropics may produce six to eight stools a day with little or no fever. The attack may last but a day or two and is considered food poisoning. Such persons may become carriers exposing others to infection thus causing epidemics. Average cases as seen in temperate climates have more or less severe diarrhea and cramps, loose or watery stools with or without blood and pus and may last from ten to fourteen days either clearing up entirely or being followed by a long continuing ulcerative colitis. Acute, fulminating cases show an abrupt onset with fevers up to 104° F., neutrophile leukocytosis or leukopenia, severe pain and tenesmus with ten to sixty discharges a day containing mucopus. There may be vomiting, abdominal tenderness, rapid dehydration, exhaustion and in severe cases death within twenty-four hours.

### Complications

Perforation may occur in the acute fulminating type. With bacteremia other parts of the body may be invaded. The anal region becomes excoriated, abscesses and fissures may develop and hemorrhoids or prolapse may be produced. Cicatricial and infiltrative changes may cause narrowing of the lumen. The acute infection may result in allergic sensitization of the mucosa producing ulcerative colitis.

### Diagnosis

The symptoms with the proctoscopic findings of diffuse hyperemia, shallow ulceration or erosions and discharge of yellow mucopus are fairly characteristic. Definite diagnosis depends upon stool examination. In two thirds of cases cultures will be positive during the first five days. By the tenth day only 10 per cent are positive. Cytologic study with the finding of pus cells, degenerated epithelial cells and microphile leukocytes is of great value. Specific bacteriophage is found in 80 per cent of cases. Agglutination tests become positive after a week. They are not as specific as the Widal reaction and may remain positive for years without continuing infection. Skin tests are in the same category.

### Treatment

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Among the many intestinal parasites described in the chapter on Parasitic Diseases (see p 134) amebic dysentery is the most serious and is often confused with ulcerative colitis of the allergic type. The whole subject of amebiasis is covered in that chapter.

### MYCOTIC DISEASES

A number of yeasts and fungi account for lesions in the intestines. Such lesions may be due to actual involvement of the lymph nodes or mucosa often confused with carcinoma and lymphoma. In other instances sensitivity to the fungi may produce allergic reactions in the intestines even typical ulcerative colitis. Mycotic diseases are discussed on page 623.

### INTESTINAL PNEUMATOSIS

This is a rare disease in which cystic accumulations of gas are present in the walls of the intestine in a manner suggesting emphysema. A large proportion of these cysts have been found in the small intestine, many in the ileocecal region, a small number in the colon and some in both small and large intestines of the same patient. They have also been found in the gallbladder and urinary tracts. The gas accumulations or pseudocysts occur singly or in clusters like grapes and may vary greatly in size from small bubbles up to 8 or 10 cm in diameter. The mucosa is intact, the gas lying between the layers of the intestinal wall, often under the serous coat. Upon puncture the gas escapes and if there is a considerable amount a pneumoperitoneum may be apparent. These pseudocysts cause no symptoms and are usually found at operation or autopsy or occasionally during an x-ray study. A few cases of obstruction of the small intestine due to pressure on the lumen have been reported. Volvulus has also been observed. The etiology is not definitely known but frequent occurrence with or after diseases or trauma involving the intestine and particularly with pyloric stenosis suggests that the gas may enter through a small breach in the mucosa and spread along the wall. Another theory is that malnutrition may be the cause. There is no infection present in the typical case.

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### Treatment

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### FISTULAS BETWEEN THE DUODENUM OR JEJUNUM AND THE TRANSVERSE COLON

In this location fistulas are serious. They may result from an ulcer perforating from the small into the large intestine or from a cancer of the transverse colon perforating into the stomach or the small intestine. Gastrojejunocolic fistula is the name given to a fistula formed by perforation of a postoperative gastrojejunal ulcer into the transverse colon.

#### Symptoms

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Figure 64 *a* Ileorectal fistula 1 Cecum 2 ileum 3 fistula 4 rectum—remainder of colon visible *b* Gastroileostomy 1 Stomach 2 gastroileostomy stoma 3 terminal ileum 4 cecum

### ILEAL FISTULAS

These are extremely rare. They usually occur between the ileum and sigmoid and usually are the result of injuries at appendectomies or pelvic operations. The diarrhea produced by such fistulas are usually not crippling, and their cause may not be discovered until careful x-ray study. During fluoroscopy, when a barium enema is being given, the sudden appearance of the barium in the terminal ileum and ascending colon when the head of the column is still in the sigmoid is characteristic, and films taken at that time will confirm the diagnosis (Fig 64 *a*). The treatment is surgical and with adequate preparation and modern techniques is almost without risk.

### GASTROILEAL FISTULAS

These are a result of an error in surgery, the ileum having been mistaken for the jejunum when a gastroenterostomy was performed (Fig 64 *b*). They are discussed under complications following surgery from ulcer.

### FISTULAS FROM THE INTESTINES

Such fistulas to the pelvis, bladder, ureter, rectum, and externally through the skin are discussed in the chapters on regional enteritis, ulcerative colitis, dysentery, tuberculosis, and cancer.

### ANAL AND RECTAL FISTULAS

These fistulas resulting from neglected ischioanal or perirectal abscesses, from birth injuries, from radium or x-ray treatments of pelvic

cancers or following operations are difficult surgical problems. They cause rectal discharges, irritative diarrhea and pelvic pain and with much induration or cicatricial contraction may cause impaction or actual obstruction. They are recognized by proctoscopy and by x-ray study (see p. 437).

#### EXTERNAL FISTULAS

External fistulas following operations upon the stomach, intestine, gall bladder, urinary bladder and pelvis are very troublesome. Aside from the annoyance caused by them, their odor and the pain they cause, they result in general debility, anorexia, insufficient food intake and the resulting symptoms of dietary insufficiency. Although occasionally such a fistula will heal spontaneously, usually surgical operation is necessary.

#### INTESTINAL NEOPLASMS

Neoplasms occur much less frequently in the small than in the large intestine. In both regions they are in general of the same type but with minor modifications in growth and symptoms. In recent years many more are being recognized, owing either to an actual increase in the incidence or to better diagnosis. No satisfactory etiological factor in the production of these tumors has been found. Irritation has been suggested but this certainly would not account for the many hereditary tumors. In the case of many intestinal neoplasms the terms "benign" and "malignant" are relative. Some benign tumors tend to become malignant, some that appear malignant behave like benign tumors.

#### BENIGN NEOPLASMS

Benign neoplasms may be considered to include all benign growths from simple epithelial hyperplasia to tumors which show a tendency to malignant degeneration. Included also could be tumors involving the intestine from without and even pancreatic rests, cysts and granulomas resulting from parasites and paraffin tumors. Rare tumors include lipomas, fibromas, neurofibromas, teratomas, hamartomas, vascular tumors, myomas and leiomyomas, the last three showing a tendency to metamorphosis into sarcoma. Carcinoids have been included among the malignant neoplasms. Because they resemble each other and usually cause similar symptoms, none of these tumors can be diagnosed definitely during life except when biopsy specimens can be obtained or when pieces of tissue are broken off and are found in the stools. In general it has been demonstrated that benign tumors occur in the colon in about 10 per cent of persons, in the small intestine in less than 1 per cent.

#### Polyps (Adenomas)

The most common tumor generally described is the *polyp*. This is a general term which includes any pedunculated growth protruding from

the surface of the mucosa. It is best and simplest to designate as polyps the common benign growths both polypoid and villous or papillary.

*Adenomas* may be small or large and may occur singly or frequently; there may be two or more in the same patient. The term *polyposis* or *adenomatosis* refers to a congenital or hereditary condition in which there are polyps close together from one end of the small intestine or colon to the other. So-called *deviant adenomas* are those which show *changes tending toward carcinoma*. In *pedunculated polyps* the tip may show carcinomatous changes first and then involvement that extends along the stalk until the whole polyp is involved and invasion of the intestinal wall takes place.

*Etiology* Various theories have been advanced as to the cause of benign tumors. Chronic irritation has been indicated; embryonal defects have been suggested; and some familial tendency to epithelial or subepithelial hyperplasia transmitted through genes has been postulated. Nothing has been established except that polyps do occur in families.

*Incidence* Adenomas of the familial type are usually discovered in the first three decades of life; the other types usually after the fourth or fifth decade. They are slightly more common in males than females. Many without symptoms are discovered on routine examination. Even those with symptoms may not be recognized as the cause for many years.

As the incidence, behavior, symptoms and signs vary somewhat according to location, those of the small and large intestine will be discussed separately.

#### POLYPS OF THE SMALL INTESTINE

In the small intestine adenomas vary in size from 1 mm. to 1 or 2 cm., rarely being of large size. Myomas, including leiomyomas, are second in frequency, occurring here more frequently than in the colon. Unless there is increase in size or a malignant change, these benign tumors may cause no symptoms. With surface sloughing or ulceration or central necrosis, hemorrhages may occur, either small and hardly noticed except for a secondary anemia of mysterious origin, or large and obvious. The latter in every way resembles massive hemorrhage from a peptic ulcer. Necrosis may also cause a perforation indistinguishable clinically from other causes of an acute abdomen. Upon growing larger, the tumors may produce an intestinal obstruction. Obstruction is also caused at times when a pedunculated polyp is carried downward by peristalsis and the intestinal wall drawn after it, causing intussusception. In this way a polyp may pull the small intestine into the cecum and up into the ascending colon, with attendant bleeding and with often extensive gangrene of the strangulated gut.

*Multiple or familial adenomatosis or polyposis* is less frequent in the small than in the large intestine. Its *symptoms* are usually those asso-

ciated with regional ileitis such as diarrheas usually bloody with abdominal pains dependent upon the amount of obstruction caused by the tumors. In some cases no symptoms may occur until complications develop when characteristic symptoms of hemorrhage perforation or obstruction may be expected.

### Diagnosis

In any patient a *history* of indefinite abdominal pains of intestinal bleeding or of an unexplained anemia is an indication for an intensive gastrointestinal study. *Physical examination* may show nothing of note except anemia until the intestinal tumors have grown large enough to be palpated or until complications have occurred such as obstruction or perforation. There is however one unexplained finding frequently encountered in polyposis of both the large and small intestine namely the occurrence of black *melanin spots* in the mucosa of the mouth and lips and occasionally of the genitals and rectum. These spots resemble freckles. They may also be seen in the skin about the nose and mouth and fingers or toes. In addition there have been reported cases in which *exotoses* dermoid cysts fibromas fibrosarcoma and leiomyoma have been found.

*Proctoscopic examination* may show nothing abnormal although the finding of rectal or sigmoid polyps should occasion a suspicion of similar tumors higher up.

### X-ray Examination

Roentgen study must be carefully carried out with films at one half to one hour intervals in different positions to attempt to show the characteristic oval defects due to polyps. Despite the greatest care and skill all but the largest polyps are usually missed. Even an intussusception is often misinterpreted. A peculiar defect may be found in the lumen of the cecum and ascending colon sometimes even in the ileum suggesting a cancer. If carefully studied the defect will show up as translucent and will reveal the characteristic markings of the small intestine lying within the lumen of the large intestine (Fig 65). This condition may be visible at one time and not at another. The defect may often be clearly seen during the gastrointestinal series and even in the gas filled intestine shown in a scout film. A barium enema may then push the small intestine and the tumor back into the ileum or higher with complete disappearance of the intussusception. The careful study may disclose the oval defect caused by the polyp in the small intestine. Cases have been reported in which the ileum was intussuscepted slowly through the entire colon finally protruding from the anus. Large polyps may cause complete

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*Figure 65* Lipomatous polyp ileum intussusception 1 Dilated ileum 2 ileal markings in cecum 3 ileal markings in ascending colon 4 ileal polyp

obstruction showing dilated intestine proximal to them (see Fig 68) The x ray findings in multiple polyposis consist in multiple defects of varying size in the mucosal outline

### Treatment

When a tumor of any kind has been demonstrated in the small intestine with or without the complications mentioned surgical intervention is indicated. In view of the tendency to malignant change and the fact that such change is often not readily discernible a wide resection of the area would be the safest procedure. When we realize that the removal of more than one third of the small intestine is not compatible with life it can be seen that multiple polyposis must be treated expectantly with removal only of areas which may be causing obstructions or in which malignancy is suspected. X ray treatments have been tried with but little success. However they may reduce or stop bleeding for a while.

### COLONIC ADENOMAS

Adenomas polypoid and papillary are much more common in the colon than in the small intestine. In careful autopsy studies in adults the incidence has been found to be from 10 to 20 per cent or more. Adenomas rarely occur singly two or more being usually found in different parts of the intestine. In routine proctoscopic examinations an incidence of 5 to 10 per cent or more has been reported. Bargen has estimated that some

form of polypoid hyperplasia of the colonic mucosa is present in 50 per cent of persons past the age of thirty years. The polypoid changes in ulcerative colitis due to hyperplasia of the multiple islands of the mucosa remaining in the areas of mucosal sloughing often called pseudopolyps must be differentiated from true adenomas. The striking feature of adenomas is their tendency to undergo malignant change as has been mentioned above. It is therefore entirely reasonable to attribute the cancer occasionally found in ulcerative colitis and diverticulosis to such adenomas already present before the disease started the malignant changes in the adenomas perhaps encouraged by repeated or prolonged irritation caused by the colitis. This offers a better explanation than that the pseudopolyps were the cause.

True multiple familial adenomatosis in which polyps of various sizes are disseminated throughout the colon is a rare hereditary disease. In these cases the tendency to malignant change may result in carcinoma occurring in two or more parts of the colon at the same time. Other tumors already mentioned in the introduction are extremely rare and usually can be identified only on microscopic sections. Any of the intestinal tumors may ulcerate, may become infected or may become necrotic with resultant sloughing, hemorrhage or even perforation. These complications are said to be more frequent in patients showing the mucosal and skin pigmentation referred to above.

### Symptoms

Small tumors may exist for years without causing symptoms. In general the symptoms depend upon the size and location of the tumors, whether they are single or multiple and how much of the colon they involve. With the development of the complications just mentioned the symptoms of hemorrhage, perforation or obstruction will call attention to the presence of some lesion as a cause. Even uncomplicated tumors may cause more or less intermittent pain or distress accentuated when the colon is active as after a meal and during or after defecation. Mild retrostaltic symptoms may occur at the same time. Occasionally bleeding, either slight with or after defecation or profuse melena may be the first hint of trouble. Acting as a foreign body, the tumor may irritate the colon, increase peristalsis and cause diarrhea with stools often though not necessarily tinged with blood. Occasionally but especially if situated in the distal colon a polyp may be pushed down by peristalsis causing an intussusception or mucosal prolapse, the tumor even protruding from the anus at times. Rarely a pedunculated tumor may break off and be expelled. When complications occur their symptoms will be prominent with the reason for a perforation or obstruction obscure until further study may reveal it.

## 376 Different Parts of the Gastrointestinal Tract

### Physical Examination

Unless a tumor of large size can be palpated or a complication has occurred physical examination is usually not of much help. Rarely a polyp can be felt on rectal digital examination either because it originates in the rectum or has been carried down by mucosal prolapse. Melanin spots as described above should be looked for.

### Proctoscopy

Tumors in the rectum and sigmoid may be shown and should be a definite indication for a complete gastrointestinal study for the reason that these lesions may occur simultaneously in any part of the gastrointestinal tract from the esophagus to the rectum. A polyp may be removed, its base thoroughly fulgurated and the specimen sent for microscopic diagnosis. Scrapings and smears from the rectum and sigmoid should be sent to an expert on cytologic diagnosis since cancer can occasionally be discovered in this way also.

### Laboratory Examination

Though the finding of occult blood in stools may indicate some bleeding from the mouth to the anus, finding it persistently in multiple tests is sufficiently suspicious to call for gastrointestinal study. Gross blood is of great significance. Smears and cultures help to rule out parasitic infestations and specific infections.

*Gastric analysis* may show bleeding from a gastric polyp occasionally associated with polyps in the intestines.

*Blood examinations* of various kinds may be indicated. The degree of anemia may be an important guide to diagnosis and therapy.

### X-ray Examination

Both a complete gastrointestinal series and barium enema study are essential. Small tumors anywhere in the gastrointestinal tract are difficult to recognize but even small colonic adenomas can be demonstrated when great care is used. A suspicious oval defect seen in twenty-four and forty-eight hour films after ingestion of barium may be identified when careful fluoroscopic examination and multiple films are made before and after evacuation of a barium enema by the double contrast method (Fig 66). The finding of a given smooth round or oval defect in the lumen at different levels in successive films indicates a pedunculated lesion. Multiple adenomatous lesions are usually also fairly well rounded but care must be exercised in differentiating them from the more irregular protrusions in the shortened and usually stiffened colon of ulcerative colitis (Fig 67).

Even *cholecystographic* studies may be of value when it is realized that adenoma of the gallbladder may occur with intestinal adenomatosis.



**Figure 66** *a* Polyp of cecum 1 Ileum 2 polyp 3 hepatic flexure Remainder of colon well seen *b* Polyp of distal transverse colon 1 Transverse colon 2 polyp 3 descending colon



**Figure 67** *a* Polyps of sigmoid 1 Polyp with narrowing 2 simpl polyp 3 dilated cecum and ascending colon 4 spasm at Cannon's ring *b* Multiple polyps = familial (barium enem) Note multiple defects throughout colon from cecum to sigmoid

In the presence of obstruction or perforation the findings are the same as for these conditions resulting from other causes

### Diagnosis

It is often difficult to differentiate between benign and malignant lesions and between intrinsic tumors and external pressure or invasion. However, as the treatment is always surgical, these errors are not serious.

### Treatment

In view of the possible errors in diagnosis and the tendency toward malignant degeneration, the finding of any tumors in the intestines calls for surgical removal. Whether simple excision or extensive resection of individual tumors is to be done depends on the surgeon's judgment. In familial multiple adenomatosis a total colectomy is usually recommended. Some surgeons feel that destruction through the proctoscope of all rectal polyps by fulguration followed by colectomy and ileoproctostomy or ileosigmoidostomy is sufficient. This permits use of the rectum for defecation, although the liquid contents from the ileum may be difficult to control at first. Such a procedure calls for subsequent frequent proctoscopies so that any new polyps may be promptly fulgurated. The usual preoperative and postoperative care of the patient must be emphasized.

### Prognosis

The immediate mortality rate resulting from bowel resections is still high, although it is gradually being reduced. Even after extensive resections there is a tendency for adenomas to recur in other parts of the intestines, with the ever present danger of malignant change. It is therefore well to give a guarded prognosis.

## MALIGNANT NEOPLASMS OF THE INTESTINES

Cancer is more frequent in the large than in the small intestine. In the small intestine, with its small lumen, it would be expected that cancer would soon cause obstruction, thus calling attention to itself. However, this is overbalanced by the fact that its contents are always liquid and normally pass rapidly through the small intestine. In the colon the contents become increasingly solid, so that obstruction calls attention to the cancer earlier in the distal colon (see *Gastrointestinal Cancer*, p. 119).

### CANCER OF THE SMALL INTESTINE

#### Pathology

Adenocarcinoma is most frequent. In the duodenum about half of the cases develop in the region of the ampulla of Vater and are called ampullary carcinoma. It is a question whether they should not be classified

is of biliary tract origin. At any point cancer probably may develop from small polyps which are also rare. Secondary involvement from the biliary tract, liver, stomach and colon may also occur. Malignant lymphomas though rare are more common in the small intestine than in the colon. The sarcomas may originate in the intestinal wall or as in Hodgkin's disease in neighboring lymph nodes often retroperitoneal. Carcinoids the yellow argentaffin tumors most frequent in the appendix occur rarely in the small intestine more commonly in the rectum and cecum. Many metastasize early and are rapidly fatal. They are now included among the precursors of cancer. A Meckel's diverticulum may undergo malignant degeneration. Spread of cancer takes place by direct extension to neighboring organs or by metastasis to regional lymph nodes to Virchow's gland and late to bones or brain.

### Diagnosis

Although theoretically a small intestinal cancer should be recognized early this is not usually the case. A careful history and thorough x-ray study should however provide the diagnosis.

### Symptoms

In a patient with no previous gastrointestinal symptoms attacks of peristaltic midabdominal pain from increased peristalsis due to irritation of the intestinal wall by the growth may occur fairly early yet are frequently overlooked. If the growth is in the duodenum the symptoms may resemble those of ulcer but will soon be followed by jaundice as the impulla becomes obstructed the symptoms then pointing to the biliary tract. In Meckel's diverticulum the cancer may set up symptoms resembling those of appendicitis. As the growth increases in size obstructive symptoms and midabdominal peristaltic pains with vomiting may occur at first intermittently and finally persistent when obstruction is complete. Intermittent pains with retrostaltic symptoms and vomiting should therefore be a signal for complete study before obstruction occurs. Though bleeding is common the changed often black blood in the stools may not be noticed. There may be pallor weakness and dizziness. With complete obstruction a marked reaction occurs with shocklike symptoms and rapid death unless prompt surgery is resorted to (see Intestinal Obstruction p. 62).

### Physical Examination

There may be little pallor or evidence of weight loss. The midabdomen may be distended with gas owing to small intestinal dilatation above an obstruction. A fairly small barely palpable mass can cause obstruction. In duodenal cancer icterus will create a suspicion of biliary tract obstruction with severe and persistent vomiting. It is important to check on

distant palpable lymph nodes With carcinoids there has been described a triad consisting of pulmonary and tricuspid valvular endocarditis with pulmonary stenosis and a blotchy flush

### Laboratory Examination

The blood will show only a secondary anemia a leukocytosis and high sedimentation rate with secondary infection an azotemia if obstruction or hemorrhage becomes severe

In duodenal cancer the *duodenal contents* may contain little or no bile, some visible blood and possibly cancer tissue or cells In any part of the small intestine a bleeding cancer will produce persistent blood in the *stools*

### X ray Examination

Films should be made at frequent (one half hour) intervals for six to eight hours after the barium meal and at different angles to dispose of overlapping of coils of intestine By thus studying the whole length of the small intestine small defects any altered mucosal pattern and localized stiffening *spasm or irregular outline and increased peristalsis* may be observed early When some obstruction begins to occur an area of dilatation of the small intestine above this may be noted With films taken in



Figure 68 Adenocarcinoma of jejunum 1 Stomach 2 dilated duodenum cap 3 jejunal obstruction with dilated small intestine proximal to it

different positions and on more than one occasion the condition can usually be differentiated from regional enteritis in which the narrow areas are of greater extent and may be multiple with "skip areas" of normal lumen between. The extensive defects and marked irritability in ileocecal tuberculosis should also be distinguishable. Lateral films will show displacement forward or actual involvement of the stomach and intestine in the presence of retroperitoneal growths. A barium enema may serve to rule out colonic especially cecal cancer. Regurgitation into the small intestine may rarely get up to the region of involvement.

In the presence of actual obstruction (see p. 66) scout films will show the typical gas-filled small intestinal pattern, the so-called ladder-like appearance with the distention confined to the midabdomen and the colon collapsed (Fig. 68). At times the distention may stop abruptly at the point of obstruction.

*Peritonoscopy* must not be done in obstruction and is of little value except in the rare lymphoma case.

Biopsy of distant lymph nodes will determine whether hope of cure can be entertained.

*Punch biopsy* of the intestine is not safe and would be of no use.

### Differential Diagnosis

In the early stages the attacks of colicky pain suggestive of biliary or renal colic may require cholecystography and pyelography to rule these out. However, biliary and renal calculi may be present also in a patient with cancer. I remember a patient who was treated for Dietl's crisis until ileal obstruction was complete. Allergic reactions in the intestine may also be present. Colonic obstruction can usually be ruled out by x-ray study, but cecal obstruction will produce the same picture as small intestinal distention.

### Treatment

Early removal of the growth with a wide margin of healthy intestine is the only treatment. Fortunately metastases occur late, facilitating successful operation. This does not apply, however, to duodenal cancer, which is so bound up with the biliary tract and pancreas that extensive operation is usually required. Such an operation is described under consideration of the pancreas (p. 608).

*Medical treatment* includes preoperative preparation by parenteral blood, nutrients and vitamins and is usually hurried because obstruction may occur early. Postoperatively the patient should be treated as after any gastrointestinal operation, that is, with early feedings and ambulation.

### CANCER OF THE COLON

Nearly 25,000 persons die of colonic cancer in this country each year. Its early diagnosis and treatment therefore present a serious public health problem. It is only slightly more frequent in the male than in the female.



distant palpable lymph nodes. With carcinoids there has been described a triad consisting of pulmonary and tricuspid valvular endocarditis with pulmonary stenosis and a blotchy flush.

### Laboratory Examination

The blood will show only a secondary anemia, a leukocytosis and high sedimentation rate with secondary infection, an azotemia if obstruction or hemorrhage becomes severe.

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Figure 68 Adenocarcinoma of jejunum. 1 Stomach. 2 dilated duodenal cap. 3 jejunal obstruction with dilated small intestine proximal to it.

lesions most frequently considered precancerous are adenomas and carcinoids. The colon is the most common site for adenomas both polypoid and papillary which may often go unrecognized for long periods of time even though bleeding is a frequent manifestation. A single polyp may be the precursor of a lone carcinoma but in multiple polyposis two or more polyps may undergo malignant degeneration at the same time. The polypoid lesions or pseudopolyps of chronic ulcerative colitis rarely have been reported as undergoing malignant changes but this has been disputed by good authority. The occasional case of carcinoma with a history of previous ulcerative colitis and the statistics from some large clinics showing such incidence suggest that the cancers developed in pre-existing adenomas as a result of irritation from the colitis. The same is true of diverticulosis. I recall a patient with colonic diverticula whose careful barium enema study revealed a small sigmoid polyp which was easily removed. Had this become malignant it would have been included among the cases showing that colonic diverticula may undergo malignant change. Extension from neighboring organs is not uncommon. Carcinoma of the pelvic organs will not only press upon but also invade the rectum and sigmoid. Gastric carcinoma may invade the transverse colon producing a gastrocolic fistula. Advanced renal carcinoma may involve the ascending colon. The lymphomas have been mentioned above.

### Symptomatology

The early symptoms occurring when the cancer is still curable are not as a rule emphasized in textbooks and unfamiliarity with these symptoms probably accounts for the frequent failure of early diagnosis. As has been emphasized in cancer in general there are no symptoms caused by the cancer as such all symptoms being due either to the mechanical effect of the growth on intestinal function or to complications. As soon as the cancer begins to grow in the wall of the colon especially if it involves the mucosa it acts as a foreign body like a particle of feces which the intestine attempts to move downward or to expel. Normal peristalsis and mucosal activity not being sufficient to expel this substance there occurs a steady increase of these activities with increasing hypermotility which may later become manifest as tenesmus and diarrhea at first only as borborygmi. The growth may also act as an irritant with resulting spasm manifesting itself as pain usually not severe at first but colicky in character. The spasm may cause retrostaltic symptoms including upper abdominal peristaltic unrest anorexia fullness sour eructations belching and in later stages vomiting. A careful history will usually reveal that these symptoms mild at first were present for many months before any definite manifestations of cancer appeared. They may have been overlooked or disregarded by the patient or if the patient had consulted his

## Pathology

Within the category of cancer of the colon are included not only carcinoma but also malignant lymphomas. *Adenocarcinoma* is the most frequent type but scirrhus and colloid carcinoma are not uncommon. Direct spread may take place by extension along the bowel wall (limitis plastica) by extension or perforation into neighboring organs and by the shedding of cancer cells from the growth. This shedding occurs after manipulation or operation and may result in implants lower down especially at points of narrowing or partial obstruction. Metastases take place not only through the lymphatics but also through the veins. The liver, lungs and bones may become involved. I have seen patients whose chief complaint was the presence of a "lump in the neck," a Virchow gland identifiable as "carcinoma probably originating in the intestine" and due to an adenocarcinoma of the ileocecal region which was causing minimal symptoms. The significance of the finding of involvement of such a distant gland must not be overlooked. It indicates that the lesion is incurable. Colonic carcinoma often goes unrecognized so long that there is opportunity for spread by direct extension or perforation to neighboring organs. The omentum, the biliary tract, pancreas, pelvic organs and even the stomach are frequently included in the mass. The severe symptoms of gastrocolic fistula due to perforation through a carcinoma of the transverse colon involving the greater curvature of the stomach may cause the patient to seek medical aid. I have also seen patients who have reached me with extensive carcinomatosis with myriads of peritoneal metastases and ascites in whom no diagnosis had been made and in whom at this stage the origin of the lesion, whether from the gastrointestinal tract, pelvis or elsewhere, could not be established.

*Sarcomas* and *Hodgkin's disease* are rare in the colon, usually originating in neighboring lymph nodes, often retroperitoneal. Sarcoma may also occur in myomas, leiomyomas and fibromas. The *leukemias*, while causing no actual intestinal lesions, may cause indefinite gastrointestinal symptoms, perhaps attributable to splenomegaly.

*Carcinoids*, "argentaffin tumors," also rare in the colon, are included under this heading because of their tendency to become malignant or to act directly like adenocarcinoma. Some are curable by excision, others metastasize extensively and are rapidly fatal. They are found mainly in the cecum, appendix and rectum and may be distinguished by their yellow color.

## Etiology

Although chronic irritation is recognized as a cause of cancer and although cancer does occur in patients with a previous history of irritation from the cathartic or enema habit or from long standing allergic irritation, these causes are not being emphasized today. The intestinal

with consequent neglect of the carcinoma a little higher up. It is well to emphasize that bleeding from any orifice requires a complete study to rule out cancer.

*Anemia* may occur as a result of the anorexia and decreased food intake but later is due to bleeding. Occasionally especially in cancer of the cecum an increasing pallor will be the first symptom noted by the patient's family and will result in a trip to the family physician. Treating a patient for anemia without knowing its cause adds further to the death rate from cancer.

*Emaciation* is much emphasized as an early symptom though it may occur early is a result of anorexia; it is frequently a late symptom and indicative of extensive involvement, bleeding, diarrhea or vomiting. When adequate amounts of food are taken and absorbed the patient may maintain his normal weight to the end even though many of these complications are present.

*Distress* is often an early symptom of complications. It may be due to an otherwise unnoticed anemia, to malnutrition in general or to reflex pressure of a pelvic mass.

*Fever* and *leukocytosis* may be present and usually indicate extensive necrosis of the growth, secondary infection or walled off perforation. I have seen such patients whose unexplained fever caused them to seek medical aid.

The symptoms of complications may be produced late or at times may constitute the first evidences of cancer. A severe hemorrhage due to sloughing off of cancer tissues may be a late symptom and a cause of sudden death or it may be the first symptom noticed by the patient. Symptoms of intestinal obstruction are frequently the first indication to the patient that anything serious is ailing him. I have seen patients enter the hospital with symptoms of an "acute abdominal calamity" due to perforation of a carcinoma with no previous suspicion of a grave illness. In one case fecal vomiting and sour smelling diarrhea due to a fistula to the stomach brought the patient to me. He had been treated for allergy. At times metastases may account for the first symptoms as in the case of the patient whose only real reason for seeing me was the discovery of a "lump" in the neck, a Virchow gland. A large abdominal mass not even recognized as such but called a "lump in the stomach" or a bad ascites due to metastases to the liver, omentum or peritoneum with the story of increasing weight with decreased food intake may bring the patient to a doctor.

### Diagnosis

Since colonic cancer is the most favorable from the standpoint of cure diagnosis in its early operable stage is most important.

physician about them he may have received a placebo for indigestion antibiotics for virus infection laxatives for constipation or astringents and opium for diarrhea. It is in cases showing such an insidious onset that a careful and complete gastrointestinal study may reveal a curable cancer.

The symptoms of *complications* are frequently the first complaints the patient describes to his physician. "Change in bowel habit" is mentioned in all textbooks as an early symptom and usually is described as constipation. As mentioned before the earliest change in habit is usually the opposite a tendency to increased frequency of or desire for defecation or even diarrhea. When this increased peristalsis is succeeded by actual partial obstruction due to increase in the size of the growth or of the area of involvement constipation will occur often alternating with diarrhea as the obstruction is temporarily relieved. Finally constipation with symptoms of intestinal obstruction will supervene.

The symptoms may vary with involvement of different parts of the colon. In the *cecum* and *ascending colon* the large lumen with liquid contents will often minimize early symptoms. Diarrhea may occur as an early symptom however accompanied by tenesmus and pain sometimes resembling appendicitis. Involvement of the *hepatic flexure* and *transverse colon* may cause pain confused with the pain of gallbladder disease or peptic ulcer. When the *splenic flexure* is involved the colicky pain may be localized in that region or may be referred to the right side. In the *descending colon* and *sigmoid* where the colonic contents are normally more solid and will not pass as easily through even a slightly narrowed lumen the pains of partial obstruction may occur earlier than in any part of the colon. These are at first intermittent as a result of an impaction which becomes liquefied or is tunneled through and are finally constant and severe. Unfortunately we often see patients for the first time at this stage.

*Bleeding* is an almost constant symptom of adenocarcinoma but occurs regularly in all forms of cancer. At first insidious and recognizable only by the finding of occult blood in feces after a meat free diet it may be overlooked by the patient even when more definite. Many persons never look at what they expel. Frequently patients may have been passing frank blood with or without mucus for considerable periods of time and will rush to a doctor when by accident they first see blood in the toilet bowl. Unfortunately it happens much too frequently that the doctor without even a rectal digital examination which might disclose the cause will diagnose hemorrhoids and prescribe an ointment or suppositories. The patient may actually have hemorrhoids due to interference with return circulation by a sigmoid or rectal cancer and be subjected to operation or injection for these with no previous proctoscopy or x ray study and

with consequent neglect of the carcinoma a little higher up. It is well to emphasize that bleeding from any orifice requires a complete study to rule out cancer.

*Anemia* may occur as a result of the anorexia and decreased food intake but later is due to bleeding. Occasionally especially in cancer of the cecum increasing pallor will be the first symptom noted by the patient's family and will result in a trip to the family physician. Treating a patient for anemia without knowing its cause adds further to the death rate from cancer.

*Emaciation* is much emphasized as an early symptom though it may occur early as a result of anorexia it is frequently a late symptom and indicative of extensive involvement bleeding diarrhea or vomiting. When adequate amounts of food are taken and absorbed the patient may maintain his normal weight to the end even though many of these complications are present.

*Dizziness* is often an early symptom of complications. It may be due to an otherwise unnoticed anemia to malnutrition in general or to reflex pressure of a pelvic mass.

*Fever and leukocytosis* may be present and usually indicate extensive necrosis of the growth secondary infection or walled off perforation. I have seen such patients whose unexplained fever caused them to seek medical aid.

The symptoms of *complications* may be produced late or at times may constitute the first evidences of cancer. A severe hemorrhage due to sloughing off of cancer tissues may be a late symptom and a cause of sudden death or it may be the first symptom noticed by the patient. Symptoms of intestinal obstruction are frequently the first indication to the patient that anything serious is ailing him. I have seen patients enter the hospital with symptoms of an "acute abdominal calamity" due to perforation of a carcinoma with no previous suspicion of a grave illness. In one case fecal vomiting and sour smelling diarrhea due to a fistula to the stomach brought the patient to me. He had been treated for allergy. At times metastases may account for the first symptoms as in the case of the patient whose only real reason for seeing me was the discovery of a "lump" in the neck a Virchow gland. A large abdominal mass not even recognized as such but called a "lump in the stomach" or a *bad ascites* due to metastases to the liver omentum or peritoneum with the story of increasing weight with decreased food intake may bring the patient to a doctor.

### Diagnosis

Since colonic cancer is the most favorable from the standpoint of cure diagnosis in its early operable stage is most important.

physician about them he may have received a placebo for "indigestion" antibiotics for "virus infection" laxatives for constipation or astringents and opium for diarrhea. It is in cases showing such an insidious onset that a careful and complete gastrointestinal study may reveal a curable cancer.

The symptoms of *complications* are frequently the first complaints the patient describes to his physician. "Change in bowel habit" is mentioned in all textbooks as an early symptom and usually is described as constipation. As mentioned before the earliest change in habit is usually the *opposite* a *tendency to increased frequency of or desire for defecation* or even diarrhea. When this increased peristalsis is succeeded by actual partial obstruction due to increase in the size of the growth or of the area of involvement constipation will occur often alternating with diarrhea as the obstruction is temporarily relieved. Finally constipation with symptoms of intestinal obstruction will supervene.

The symptoms may vary with involvement of different parts of the colon. In the *cecum* and *ascending colon* the large lumen with liquid contents will often minimize early symptoms. Diarrhea may occur as an early symptom however accompanied by tenesmus and pain sometimes resembling appendicitis. Involvement of the *hepatic flexure* and *transverse colon* may cause pain confused with the pain of gallbladder disease or peptic ulcer. When the *splenic flexure* is involved the colicky pain may be localized in that region or may be referred to the right side. In the *descending colon* and *sigmoid* where the colonic contents are normally more solid and will not pass as easily through even a slightly narrowed lumen the pains of partial obstruction may occur earlier than in any part of the colon. These are at first intermittent as a result of an impaction which becomes liquefied or is tunneled through and are finally constant and severe. Unfortunately we often see patients for the first time at this stage.

*Bleeding* is an almost constant symptom of adenocarcinoma but occurs regularly in all forms of cancer. At first insidious and recognizable only by the finding of occult blood in feces after a meat free diet it may be overlooked by the patient even when more definite. Many persons never look at what they expel. Frequently patients may have been passing frank blood with or without mucus for considerable periods of time and will rush to a doctor when by accident they first see blood in the toilet bowl. Unfortunately it happens much too frequently that the doctor without even a rectal digital examination which might disclose the cause will diagnose hemorrhoids and prescribe an ointment or suppositories. The patient may actually have hemorrhoids due to interference with return circulation by a sigmoid or rectal cancer and be subjected to operation or injection for these with no previous proctoscopy or x-ray study and

### Laboratory Procedures

Among other laboratory procedures examination of *stools* for blood is much stressed. Visible blood of course is of great diagnostic value but the finding of occult blood even though the patient has been on an absolutely meat free diet for three or four days or longer before the stool is collected is subject to so many errors that it cannot really be accepted as the only indication of possible malignancy. However if its finding results in a careful gastrointestinal study to rule out cancer some good will have been accomplished by the test for it. Cytologic examinations of stools and cultures and microscopic examination of what appear to be particles of tissue may be of value. The material obtained by proctoscopy or by means of a colonic brush pushed upward through the proctoscope preferably under fluoroscopic control being fresher and not as much contaminated is of much greater value.

Examination of *gastric contents* may give little information except when the stomach is secondarily involved in which case they may show blood malignant cells or even feces.

*Blood studies* are of no specific value but will show a secondary anemia at times before other evidences of bleeding have been observed. In the presence of secondary infection or with extensive necrosis of a tumor a leukocytosis may be of help as may also a prolonged sedimentation rate. A routine serologic study may at times disclose syphilis as a possible cause of the symptoms but will not rule out cancer.

The *urine* will show no characteristic findings unless the urinary tract has become involved in the growth. Every once in a while some urine test such as the methylene blue test is brought forward as a specific test for malignancy but is later abandoned. Unfortunately as yet no specific test indicating the presence of malignancy has been evolved.

*Biopsy* of apparently involved glands in the inguinal region or in the supraclavicular space will establish a diagnosis of late incurable malignancy.

### X ray Examination

X ray study by an expert roentgenologist is the most important aid to diagnosis. It will almost invariably show early lesions of the colon except in the rectum which is not only difficult to visualize adequately but which can be better explored with the proctoscope and biopsy for definite diagnosis (see p 447). In general involvement of the colonic wall will manifest itself (1) as an area of defect or merely of stiffening due to induration (2) as an irregular area or multiple small areas of protrusion of barium into ulcerations (3) as localized spasms if lesions are small (4) as an annular spool like narrowed area or (5) in later stages through evidences of obstruction or perforation.

The x ray study should be complete and detailed with repeated exam



### History

This should be complete. Hereditary factors should be considered. A careful past personal history is important. The gastrointestinal history should include in detail the eating habits of the patient, the bowel habit, the kind of laxatives used if any, the frequency with which they were taken and their effect. The occurrence of such symptoms as previously mentioned, especially in a patient with no previous gastrointestinal disturbance, must be carefully and tactfully searched for. The clinician must not wait for the textbook symptoms of pain, emaciation, pallor, or hemorrhage to suspect cancer. It is the mild, often indefinite symptoms which are important.

### Physical Examination

In a truly early case there should be no physical signs indicating cancer. The palpation of a tumor, the observance of distention, the hearing or seeing of peristalsis, the finding of an enlarged liver, metastatic glands, and ascites are all so late in occurrence as to be only of academic interest. A rectal digital examination may at times disclose an early cancer or a precancerous polypoid lesion. More than half of rectal carcinomas are within reach of the examining finger, especially if the patient is examined in a squatting position. Hemorrhoids, especially if of recent origin, can be caused by a cancer higher up, and their discovery, especially in older patients, calls for careful proctoscopy and a thorough x-ray study.

### Proctoscopy

Proctoscopy, with biopsy of a rectal or sigmoid lesion, can determine the diagnosis if the specimen is removed from the firm, hard periphery of the ulcerated, indurated area and not from a sloughing area at its center. A polyp may be removed in its entirety, preferably by means of electrocoagulation of its pedicle, and submitted to microscopic study. I have seen a sigmoid polyp with malignant degeneration presenting itself just above the internal sphincter because it had caused an intussusception or mucosal prolapse. The polyp could be felt on simple digital examination. The discharge from an ulcerating lesion may be simply blood, a grumous material mixed with mucus, or if infected, it may contain pus. An immediate fresh smear of this material, even if it shows *Entamoeba histolytica*, does not necessarily rule out coexisting malignancy. The rectal discharge or material obtained from swabs may be subjected to cytologic study by the Papanicolaou technique and at times will be of help in making a diagnosis when the lesion is too high up for removal of a biopsy specimen. Cultures are indicated, not only to rule out specific dysentery organisms, but also to determine the specific cause of secondary infection if this is present.

and subsequent films taken with the colon filled and after evacuation may show the ileum filled sufficiently to rule out defects due to disease. Attempts at filling the entire colon should not be abandoned because of a patient's lack of cooperation or the appearance of excessive spasms especially at the splenic or hepatic flexures with forcible efforts at expulsion. Even if rectal tube and barium mixture are expelled a repetition of the study is definitely indicated either at once perhaps with the aid of sedation or a short time thereafter following further preparation. I have observed tragic consequences when a barium enema study was abandoned in disgust and subsequently the patient succumbed to a carcinoma at the splenic or hepatic flexure the two colonic regions most apt to be overlooked especially when films fail to show the entire flexure. I have seen rectal cancer similarly overlooked.

Films of the completely filled colon taken at different angles to bring out regions hidden by loops at the flexures or elsewhere must be carefully studied to find areas of defect narrowing or spasm with a tendency to dilatation behind them (see Fig. 69). Multiple smooth defects due to polyposis and multiple protrusions due to diverticulosis must not be allowed to divert attention from a careful search for irregular defective areas indicating malignant changes. The total filling of the colon may cause such a dense shadow that defects may be obscured making the postevacuation films often more valuable in arriving at a diagnosis (see Fig. 70). Spasms defective areas polyps diverticula and accumulation



Figure 63. *a* Carcinoma of sigmoid nonobstructive Barium enema film in lateral position. Arrow points to annular defect. *b* Carcinoma of sigmoid nonobstructive annular. Regular barium enema film. Arrow points to irregular defective area.

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inations when any uncertain findings are observed. If a colonic lesion possibly obstructive is suspected it is best to do the barium enema study first in order to determine whether there is sufficient lumen for the gastrointestinal series. A complete gastrointestinal series with films taken at hourly or half hourly intervals for six hours followed by twenty four and forty eight hour films may be of great value in showing also other lesions of the gastrointestinal tract. In the latter part of the first six hours of the gastrointestinal series filling of the cecum may be interfered with or an irregular filling may be demonstrable. As in tuberculosis cecal irritability may be observed but as the barium column advances in the twenty four and forty eight hour films barium may often be seen adherent to irregular defective areas. This is also true of lesions in the remainder of the colon.

As involvement increases and some actual obstruction supervenes this can be demonstrated by the presence of a localized area of narrowing ringlike in the case of annular carcinoma. Dilatations proximal to such an area may indicate the site of the lesion. It must be remembered however that spasm of the sphincter at Cannon's ring at the junction of the first and second thirds of the transverse colon is frequently due to reflex or retrostaltic stimuli from obstruction in the sigmoid or rectum and that it may produce a definite dilatation proximal to Cannon's ring. This may be mistaken for and diagnosed as due to obstruction of the transverse colon at that point. A barium enema study in such a case may show a dilated rectum up to the point of obstruction (Fig 7 p 66).

*Barium Enema* Barium enema study should be done only after adequate preparation. Defects due to feces in the lumen or adherent to mucosa are difficult or often impossible to differentiate from organic involvement. The best preparation is the cleaning out of the intestine with castor oil 1<sup>1</sup> to 2 ounces taken the night before the examination and occasionally followed by soapsuds or saline enemas in the morning if no satisfactory cleaning out was accomplished. When fecal impaction is present softening of the fecal mass by rectal instillations of 6 or 8 ounces of equal parts of mineral oil and castor oil on the day or evening preceding the examination or milk and molasses enemas may be tried the castor oil being given as usual. Instrumental or digital removal of impactions may be necessary.

*Fluoroscopy* during injection of the barium mixture should be most carefully and unhurriedly carried out. The head of the barium column entering the rectum and sigmoid frequently can be seen to surround a mass or pass through a narrow point or cause relaxation of a spasm. Defects caused by neoplasms or polyps can be observed. When the barium column reaches the cecum failure of regurgitation into the ileum may indicate a lesion there or may be due to temporary spasm.

of perforation I have seen one case of rupture of the cecum due to persistent spasm of the sphincter muscle at Cannon's ring secondary to sigmoid carcinoma

A clinical aid for differentiating between spasm and partial obstruction is to feed the patient a diet containing a large residue like the anti constipation diet (see p 323) which will usually relieve simple spasm In organic obstruction within two or three days it may induce obstructive symptoms which when not relieved by enemas may call for early operation This procedure is risky and must be carefully checked

### Peritoneoscopy

Peritoneoscopy permits direct observation of the abdominal organs and of tumors and metastases and also the removal of specimens for biopsy It is particularly valuable when there can be felt in the abdomen large masses which are probably inoperable The nature of the tumor being determined by biopsy will indicate the method of treatment to be used as for instance the choice of radiotherapy for a malignant lymphoma

### Punch Biopsy

Punch biopsy which is being successfully used in liver diagnosis may at times be used to obtain specimens from other solid tumors but is attended by considerable risk

### Specific Treatment

The treatment of cancer still consists in its extirpation or destruction For cancer in general some progress has been made in the direction of treatment by hormones mainly those of the gonads by deep x ray therapy and by radioactive isotopes but thus far no such treatment of colonic cancer has proved to be successful Both extirpation and destruction of cancer produce cures only if distant areas are not involved This is true not only of carcinoma but also of the malignant lymphomas

*Extirpation* Extirpation consists in radical surgical excision not only of the growth itself but also of enough tissues surrounding it to assure removal of all metastases and extensions of the growth Surgery has reached the point today at which the mere fact that a colonic cancer has invaded neighboring organs does not deter the surgeon from removing with the growth at one operation the stomach parts of the pancreas the spleen and the omentum or in the pelvic carcinoma not only the rectum and sigmoid but also the bladder and all the pelvic organs In the absence of such spread and with only a few neighboring lymph nodes involved wide resection offers an excellent hope of cure When such resection can be combined with an anastomosis to re establish the continuity of the colon the patient can be said to be really cured Yet even

ful. However, delay in the use of real narcotics serves no useful purpose to either the patient or family. To permit a patient to suffer severe pain just to keep him conscious is inexcusable. Demerol, Pantopon and other opium derivatives are expensive and are no more desirable than morphine which at first by mouth and later parenterally should be given in adequate and increasing doses to keep the patient either comatose or at least so somnolent that there is no realization that he is dying. I had one patient who received up to 5 grains of morphine every three hours remaining conscious *without pain* to the end. At times suppositories of opium in increasing doses and frequency, starting with 1 grain every six hours, can be used to obviate the need for hypodermic medication, especially when the patient cannot afford the services of a nurse.

I have frequently found it extremely difficult to get the family to cooperate fully in the program of narcosis. I always explain to the family that these drugs were made for such patients and that they should be used to the fullest extent necessary to ease the patient's last days. The remembrance or the hearsay evidence of a cancer patient dying in great agony and the accompanying fear of being told that one has cancer is a principal deterrent to seeking medical aid when symptoms occur at a stage when cancer might be curable.

#### INTESTINAL ENDOMETRIOSIS

In endometriosis of the uterus and adnexa the lesion may spread to neighboring organs or cells or may escape from the confines of the pelvic organs and become implanted elsewhere. The most common site for these extensions and implants is the rectum, mostly in the rectovaginal septum. From contiguity with the uterus and adnexa, implants may also occur in the sigmoid, ileocecal region, appendix and in the small intestine. A few cases have been reported as occurring in the gallbladder. The tissue resembles endometrium both in structure and in function. At the menstrual cycle it becomes engorged and may bleed, returning to its previous state during the intermenstrual period. Blood may become encysted, producing nodules of varying size and density, often bluish in color. In the small intestine and appendix the lesion grossly resembles carcinoid. Usually the serosa is involved, the mucosa being intact with no direct extension into the lumen except at times in the rectal wall. A real intestinal obstruction is therefore rarely produced, although adhesions to the area of involvement may form kinks or temporary partial obstruction at the menstrual periods. The original pelvic lesion is most frequently discovered at the time of a pelvic operation, usually hysterectomy, performed supposedly for chronic pelvic inflammatory disease or for fibroids. Malignant degeneration has never been reported. Occurring as it does, mostly in women during the fourth and fifth decades of life, the onset of intestinal symptoms often leads to the suspicion of

hemorrhages occur vitamin K may be indicated. Reduction of ovarian function will usually cause diminution in size and sometimes even disappearance of the lesion. Methods used to reduce or destroy ovarian function are (1) the administration of male sex hormone one of the methyltestosterone preparations under the tongue or by mouth (2) x ray or radium therapy over the ovaries which may be used in younger patients because later ovarian function may at times be restored. On occasion x ray is combined with the hormone therapy (3) Castration the removal of both ovaries preferably with hysterectomy will frequently in an older patient clear up the secondary lesions elsewhere thus avoiding operations for these conditions.

### Prognosis

As endometriosis has not been known to undergo malignant change it is worth while trying to check it by the means suggested leaving surgery as a last resort. Once the disease has cleared up the principal danger of a recurrence lies in the subsequent careless use of estrogenic hormones.

### INTESTINAL MELANOSIS

The deposition of the pigment melanin in the submucosa of the colon and rectum and occasionally in the ileum and appendix giving the mucosa the appearance of alligator skin is unusual and may be frightening if not understood. The discoloration may be brown or black and may occur in streaks or spots. The pigment is the same as occurs in the skin and hair in Addison's disease and in melanomas. Its origin is obscure but among other theories it is supposed to be a product of bilirubin, hemosiderin or aromatic protein decomposition products. The degree of pigmentation varies in different parts of the intestine in different persons usually being found in portions of the intestines where stasis occurs normally as in the rectum and sigmoid. It may be found at any age but is most frequent in middle aged constipated people especially in those with the laxative habit. The theory that it is caused by laxatives of the irritating anthracene type rather than by the constipation for which these were taken seems to be confirmed by the greatly diminished occurrence of the phenomenon since such laxatives are not used so frequently as formerly. These drugs include cascara, senna, aloes, frangula and rhubarb either alone or in combination in the form of laxative pills. They were consumed in large quantities until more recently displaced by mineral oil, various colloid substances and mild salts. It appeared to require a year or more of continuous use to produce the pigmentation and nearly as long before it would clear up after the drugs had been discontinued. No specific symptoms were ever attributed to the pigmentation and no serious sequelae were observed although at one time

cancer the true nature of the lesion being discovered only at the time of operation. It has been found however at all ages from childhood to old age.

### Symptoms

The primary symptoms are pelvic, with intestinal symptoms secondary to them. Both occur characteristically at the menstrual period. The former consist usually in dysmenorrhea with lumbosacral backache, low abdominal and pelvic pain, dyspareunia and urinary symptoms. With involvement of the rectovaginal septum, rectal pains, tenesmus and rarely rectal bleeding may occur. Sigmoid implants may cause tenesmus and constipation or at times diarrhea. Appendiceal or ileocecal involvement will simulate attacks of comenstrual appendicitis and appendectomy is often performed only to disclose the unsuspected lesion. In the small intestine indefinite mild obstructive and retrostaltic symptoms occur. Unfortunately many of these patients are treated as neurotics, the symptoms being attributed to premenstrual tension, poor sexual adjustment or worry about the sterility frequently resulting from the condition. The use of follicular hormones aggravates the symptoms.

### Diagnosis

Any vague symptoms associated with the menstrual cycle should lead to a suspicion of endometriosis as the cause. General physical examination may reveal nothing definite except in cases in which the findings will suggest appendicitis or intestinal obstruction. Rectal digital examination will usually disclose the nodular infiltration of the rectovaginal septum.

Proctoscopy usually fails to show any sign of the infiltration except at times a slight smooth bulging and rarely a tendency to bleed. Occasionally adhesions may prevent the passage of the proctoscope.

X-rays are usually of little if any help, showing at most and rarely what resembles an extrinsic pressure defect.

Laboratory tests show nothing characteristic.

Biopsy specimens taken from the rectovaginal septum, best accomplished through the vagina, should provide the diagnosis. In other locations only operation and microscopic examination will disclose the nature of the lesion.

### Treatment

In cases of simulated appendicitis or intestinal obstruction surgery is usually resorted to, the lesion if found may not be identified and is excised. On suspicion of malignancy more or less radical resections may be performed. When the nature of the lesion is established in advance it is usually not necessary to operate except for obstruction. When

ness and muscle guarding or rigidity and the feeling of a mass. In the ileocecal region the findings simulate those of appendicitis. With perforation into the pancreas a hardened tender pancreas may be palpable. In the rectum and sigmoid foreign bodies may be found by digital or proctoscopic examination. They may cause fecal impaction and bleeding.

### X-ray Examination

When the foreign body is opaque scout films will be of help. With non-opaque bodies such as toothpicks and possibly small bones diagnosis must depend upon suspicion when the barium meal reveals deformities from adhesions or localized dilatation from partial obstructions. Perforation may disclose free air in the peritoneal cavity. Obstruction will show the usual findings.

### Treatment

*Prophylaxis* is important. As toothpicks are getting to be a frequent cause of perforation great care should be exercised to avoid swallowing those used for club sandwiches, canapes or meatballs. Two factors which favor swallowing these rough indigestible sharp objects are the drinking of alcoholic beverages and the wearing of upper dentures. Foreign bodies known to have been swallowed should be followed by x-ray study on their course through the gastrointestinal tract. To prevent them from getting blocked a probable aid is the old method of frequent feeding of foods furnishing bulk such as green vegetables, fruits and whole grains. When they have become impacted or have perforated operation must be resorted to. For such objects in the duodenum it is desirable not to open the duodenum if possible in order to avoid the danger of infection, adhesions or obstruction. With the abdomen open it is sometimes possible to milk them back into the stomach. They can then be removed through a gastroscope or even an esophagoscope under direct guidance or by means of a gastrotomy. Rectal and sigmoid foreign bodies can at times be removed by manipulation or through the proctoscope but often require surgical removal.

### Prognosis

When foreign bodies have caused damage of any kind the consequences may be serious. Aside from infection, infiltration, adhesions and obstruction, ulcers, granulomas or even cancer may result.

### INTESTINAL TRAUMA

Injuries to the intestine from external trauma are rare. They are of three kinds:

1. The trauma may be direct as from penetrating wounds or bullets usually causing one or two perforations of the bowel wall and producing



it was suggested that melanosis might predispose the bowel to cancer. The *diagnosis* has usually been made only on a routine proctoscopic examination in a patient taking laxatives for chronic constipation. Rarely it has been observed at operation or autopsy. No *treatment* except with drawal of the causative drug and the establishment of a normal bowel habit by physiologic measures is required.

#### FOREIGN BODIES IN THE INTESTINES

Most foreign bodies which pass through the pylorus have no trouble going down through the small intestine although some large ones may be delayed at the narrower ileocecal region or may even cause obstruction there. Occasionally a bezoar may protrude from the stomach into the duodenum causing pyloric obstruction. Long narrow objects such as pins, thin bones or toothpicks are usually carried through lengthwise without damage. They may enter and even perforate diverticula including a Meckel's diverticulum. In the duodenal loop they may get caught and perforate the bowel wall. As a rule such a perforation does not leak the object filling the small opening. An exudate occurs, peritoneal irritation and infection cause adhesions, the duodenum becoming deformed, splinted and dilated behind the narrowing. Occasionally the sharp point of the object may perforate the pancreas. I have seen a pin do this setting up an inflammation there (see Fig. 52, p. 298). Rarely foreign bodies may enter the intestine from the outside by perforating the wall. Gallstones may enter in this way as may necrotic bones and bullets. Large gallstones are the most frequent cause of obstruction at the ileocecal valve causing gallstone ileus. Some foreign bodies may lodge in the cecum, small ones such as pins, toothpicks or seeds may enter the appendix or colonic diverticula producing symptoms and perhaps complications of appendicitis or diverticulitis. The next points of delay are usually at the flexures or the sigmoid and rectum (see under Rectum Foreign Bodies, p. 454).

#### Symptoms

Most foreign bodies cause no symptoms in their passage through the intestines. When they cause injury or obstruction the symptoms do not differ from those due to other causes except that a history of swallowing a pin, a large pit, coin or other foreign body, or of a preceding acute episode associated with the perforation of a gallstone may occasionally be obtained. When the foreign body perforates the intestinal wall it produces the symptoms of an "acute abdomen." Injury from trauma by the foreign body will cause hemorrhage at times profuse.

#### Physical Examination

In complicated cases the abdominal findings of intestinal obstruction or perforation will be present. Localized peritonitis will produce tender

help in expelling contents. Lymphoid follicles protruding from the submucosa through the mucosa occupy about 30 per cent of the mucosal surface. The lymph nodes of Lieberkuhn line the mucosa. These large amounts of lymphoid tissue have given the appendix the name of the "abdominal tonsil." The muscular coat is in two layers, longitudinal and circular, the former shortening and steadying the appendix while the latter contracts and empties its contents. Some have described a sphincter like arrangement of muscular fibers at the junction with the cecum. Two thirds of the appendix is covered by peritoneum which has its own mesentery. Its blood supply is poor.

### PHYSIOLOGY

It has been held that the appendix has no function, being only a vestigial remnant. Its lymphoid tissue predominance has suggested that it may have a protective function against infection, as in the case of the tonsils. To its secretion, very small in amount but rich in mucin, has been attributed a lubricating action on colonic contents. It may be that with careful study, by means of radioisotopes, one or all of these functions may be proved.

It is suggestive that anatomically the valve, the sphincter, the muscular layer, and possibly the cilia, are all designed to prevent materials from entering the appendix from the cecum or to expel them if they enter.



Figure 71. Various positions occupied by the appendix (Deaver). Order of frequency: 1, to or into the pelvis (4 to 5 o'clock); 2, toward the spleen (2 to 3 o'clock); 3, behind the cecum (9 to 12 o'clock); 4, under the cecum (7 to 8 o'clock). (Bockus, *Gastroenterology*, Vol. 11.)

more or less peritonitis. In such cases symptoms of an acute abdomen are present and scout films of the abdomen will show free air in the peritoneal cavity. Supportive therapy and immediate operation are indicated.

2. A deep crushing injury to the abdomen is encountered in automobile accidents may cause rupture of various organs including the intestine, rectum and appendix especially when filled with food, feces or gas. When of such severity the injury will usually cause death. If not an attempt might be made to close the rents in the intestine but infection with peritonitis will almost surely cause death later.

3. Indirect injury with rupture of the intestines and other organs usually occurs because of a fall from a height. This is also usually fatal either at once or later as a result of severe peritonitis.

## The Appendix

### General Discussion

Although so small and with no well defined function this small diverticulum like structure by some considered to be a vestigial remnant assumes great importance because acute appendicitis remains one of the greatest killers.

### ANATOMY

The normal vermiform appendix may vary in length from  $\frac{1}{4}$  inch to  $9\frac{1}{2}$  inches in length and from  $\frac{1}{4}$  to 1 inch in diameter. It hangs from the cecum pointing downward toward the iliac fossa backward or upward behind the cecum or to either side (Fig. 71). The lumen of the appendix may appear merely as a funnel shaped downward extension of the cecum with no sharp outline the fetal appendix or may come off similarly but with a sharp definition between it and the cecum or it may as in most cases originate somewhat medially and posteriorly.

The wall of the appendix resembles that of the colon except for its large amount of lymphoid tissue. The lumen is very small. The mucosal layer is puckered and near the cecum is thrown into one or more folds. One fold the valve of Gerlach found in 80 per cent of appendices tends to prevent cecal content from entering the appendix but normally does not prevent appendiceal content from being expelled into the cecum. Some anatomists have found that the mucosa is at times everted to

the appendix. Similar defects should be looked for in barium enema and cholecystographic studies.

### Treatment

In the absence of symptoms mere displacement does not call for any treatment. With only mild symptoms a complete study must be made to rule out other possible causes. In an acute process prompt operation is of course indicated.

## Diseases of the Appendix

### DIVERTICULOSIS

Diverticulosis of the appendix is rare. Diverticula usually occur in the distal half of the appendix along the mesenteric border. They may occur alone or in association with colonic diverticula, are usually small and may be single or multiple. Causing no specific symptoms, they are rarely recognized unless acutely inflamed, when their thin walls tend to rupture easily. They are usually discovered in the appendix only after removal.

### INTUSSUSCEPTION

Intussusception of the base of the appendix into the cecum is another rare condition. It occurs as a result of a tumor in the appendix or is a part of an ileocecal or cecocolic intussusception. It is practically never recognized, the diagnosis being made of an ileocecal invagination if x-ray studies happen to be made. It is found at operation for intestinal obstruction.

### APPENDICITIS

Appendicitis is the most important and most serious appendiceal condition. Deaths from appendicitis still rank high in mortality statistics. In children under twenty-one it has consistently ranked among the top five causes of death. It occurs at all ages and is particularly dangerous in the aged.

### Pathology

Though pathologists and surgeons are not in agreement on the pathology of appendicitis, it is most convenient to discuss it as a disease which occurs in stages.

The first stage, in which only the mucosa is involved, is called *acute catarrhal appendicitis*. The mucosa shows hyperemia and edema with round cell infiltration, often eosinophilic, and with an exudate in the lumen. This corresponds to an allergic reaction and may subside quickly. If the lumen becomes obstructed from any cause and secretion continues without infection, a mucocele is formed. This cystic dilatation stretches

## Different Parts of the Gastrointestinal Tract

### *Anomalies and Displacements of the Appendix*

#### ANOMALIES

Rarely there may be a congenital absence of the appendix. The appendix may show a septum producing the "split appendix" or there may be two appendices. In visceral transposition the cecum and appendix lie on the left side.

#### DISPLACEMENTS

The appendix, normally movable and lying in various positions, may be displaced to any part of the abdomen. In case of an undescended cecum it may lie over the liver. A long retrocecal appendix may extend up under the liver and may become adherent to the gallbladder. With a low cecum or owing to its length the appendix may lie deep in the pelvis and become adherent there. Occasionally an appendix is found in the sac of an inguinal or femoral hernia.

#### Symptoms

Anomalies and displacements in themselves may cause no symptoms but with disease of the appendix they may be important to consider. Removing one of two appendices may prove embarrassing. Acute appendiceal abscess under the liver may be mistaken for acute cholecystitis or liver abscess. A retrocecal appendicitis is often overlooked being called food poisoning because of the diarrhea and vomiting it causes. An inflamed pelvic appendix may be mistaken for salpingitis or ectopic gestation.

#### Physical Examination

It is usually difficult or even impossible in an emergency to make a definite diagnosis of displacement. Tenderness and rigidity over the appendix will be attributed to the organ lying nearby. In the retrocecal type tenderness may be entirely posterior with no anterior rectus muscle spasm. Rectal examination will often disclose a mass or abscess on the right side. The pelvic appendix is not easily identifiable in an acute process.

#### Laboratory Examinations

The laboratory findings in the acute case will be the same as in any acute appendicitis (see *Acute Conditions of the Abdomen* p. 68).

#### X-ray Examinations

When no emergency exists x-ray studies twenty-four or forty-eight hours after a barium meal may show the appendix in its abnormal position or may show peculiar pressure defects in the region occupied by

catarrhal appendicitis. This conception would explain the occurrence of the disease with tonsillitis, the allergen in such a case being either the bacteria, their products or the substance produced by the breaking down of the infected tissues. It would also explain the appendicitis associated with arthritis and with acute exanthems.

The swelling of the lymphoid tissue in and around the appendix has been suggested as being at times a part of a general lymphadenopathy. The appendicitis associated with abdominal lymphadenitis in children is an example.

The most favored etiological factor is *obstruction to the lumen*. Experimentally, appendicitis produced by an allergic reaction, by fecaliths, by foreign bodies, by trauma, by swelling of lymphoid tissue, by neoplasms, by displacements, by adhesions and kinking, by strictures from previous ulceration or by any other factors are all included under this general classification. The mechanism is described as an increased peristalsis of the appendiceal musculature in an effort to overcome the obstruction, increasing dilatation of the lumen with thinning out of the wall and interference with circulation, followed by small necroses with ulceration, secondary infection and the whole picture of an appendicitis. Spontaneous subsidence is considered to be due to expulsion of a plug causing obstruction or a sudden straightening out with opening of the lumen.

It is evident that considering trauma as the invariable cause of appendicitis appears greatly to simplify the whole problem, but the many other causes of obstruction still remain as important etiological factors. In addition, sex plays a part, four times as many males as females being affected. The general condition of the patient, the previous history, as to habits of diet and general hygiene, of prompt and efficient treatment of previous diseases, and of the avoidance or removal of focal infections are all important influences on the patient's susceptibility to any infection. Even heredity plays a part, as the frequency of acute appendicitis in certain families will attest.

The diagnosis and treatment of acute and chronic appendicitis will be considered separately.

#### ACUTE APPENDICITIS

##### Diagnosis

Acute appendicitis is discussed in some detail in the chapter on Acute Conditions of the Abdomen (p. 68). It is well to remember that this serious disease is often overlooked or mistreated. Although acute catarrhal appendicitis may subside within eighteen to thirty-six hours, there is no way of knowing which case will subside or which will go on to suppuration and perforation. No laboratory methods, no x-ray examinations, no "trick hunches" will predict the outcome. In the absence of

and thus the wall of the appendix rarely forming a tumor of considerable size. If secondarily infected the thin wall is liable to perforate. If it does not subside an acute diffuse inflammatory process may follow as a result of secondary infection. This has been called *acute suppurative appendicitis* as well as phlegmonous ulcerative necrotic gangrenous or perforative appendicitis depending upon its most salient characteristic.

The first stages may or may not be present the appendix sometimes immediately becoming suppurative. In all cases there is a protective reaction a fibrinous exudate surrounding the appendix together with lymph node enlargement a *periappendicitis* or perityphlitis. This is for the purpose of walling off the infection. When the appendix ruptures this protective barrier may prevent a general peritonitis so that a localized appendiceal abscess may be formed. In a violent case the appendix may rupture directly into the peritoneal cavity, producing a general peritonitis or a neglected abscess may rupture secondarily with the same result. Secondary infections elsewhere may occur, the most common being pylephlebitis and liver abscess or pelvic abscess.

### Sequelae

When the inflammatory process subsides there remains a thickening or fibrosis of the wall of the appendix with more or less narrowing of the lumen which may contain small masses of inspissated mucus and feces called *fecaliths*. These may become infiltrated with calcium. Adhesions to contiguous structures due to the peritoneal irritation may cause kinks in the ileum deformities and angulations of the cecum deformities and constrictions of the ureter and various deformities in the pelvic organs. An abnormally placed appendix may cause adhesions to the gallbladder liver transverse colon and even the rectum. Such persistent lesions are grouped together under the convenient designation of chronic appendicitis.

### Etiology

There is still much difference of opinion over the cause of appendicitis. The observation that acute appendicitis frequently follows an acute tonsillitis tonsillectomy and upper respiratory tract infection and the fact that infected tonsils are invariably found in patients who have so called chronic appendicitis has suggested the theory that hematogenous infection is a cause. Blood cultures however have never shown any organisms in such cases. Direct infection from contiguous organs such as the ileum and cecum and the genitourinary tract especially the adnexa is an obvious cause.

That appendicitis is due to an allergic reaction is suggested by the nature of the pathological findings and their rapid subsidence in acute

The *pathology* of chronic appendicitis has been discussed above and consists of two factors the chronic changes taking place in the appendix itself including stiffening fibrosis and formation of a mucocele and the changes in its contiguous structures periappendicitis and adhesions. Recurrent attacks of acute appendicitis are also described as chronic appendicitis. Most of these chronic conditions are usually considered to be the result of an acute appendicitis.

The *etiology* has been discussed and is important from the standpoint of treatment.

### Symptoms

There may be no symptoms at all or there may be recurrent attacks of acute appendicitis with no symptoms between attacks. Manifestations of the coexisting disease may mask any symptoms pointing to the appendicitis. On the other hand the appendix region may be the area in which symptoms of the other disease are located. Peptic ulcer may produce hunger pains confined to this region gallbladder pains may be referred to the right lower quadrant and the pains and tenderness of ulcerative colitis and enteritis may be located at McBurney's point.

*General Symptoms* In the average case if symptoms are present they are usually retrostaltic identical with those due to other causes. Pylorospasm and even cardiospasm have been attributed to appendicitis. As a result of such symptoms the patient may have anorexia fail to eat properly lose weight and strength and become neurotic. Hematemesis has been mentioned as a symptom but undoubtedly it has been due to an overlooked coexistent peptic ulcer. The bowel may become irritated with resulting spasm of the colon and constipation or diarrhea also usually due to a coexistent lesion.

*Local Symptoms* There may be no symptoms in the region of the appendix. Pain in the right lower quadrant either constant or intermittent should occasion a suspicion of appendicitis but may be due to any of the many causes previously discussed. The pain may radiate. Adhesions to neighboring organs cause it to radiate into the pelvis from adnexal adhesions down the leg from psoas adhesions to the loin with colic from ureteral adhesions and to the midabdomen where it is often colicky and suggestive of obstruction from ileal adhesions and kinks. The pain may be made worse by exertion by posture by constipation or by diarrhea. It may be initiated by some article in the diet suggesting allergy.

### Physical Examination

A complete examination is essential. The patient's general condition may be below par or may not be affected. Focal infections in the mouth nose and throat pelvis rectum and urinary tract should be sought for.



all the typical findings such as the history of crampy pain nausea vomiting and the finding of tenderness fever and leukocytosis there may still be acute appendicitis. At times only a diagnosis of "acute abdomen" can be made. A single intramuscular injection of 10 or 12 minims of 1:1000 epinephrine solution will do no harm and will at times cause a complete and rapid clearing up of an acute allergic reaction. The abdomen the back the pelvis and the rectum should be carefully examined for tenderness or mass. At times patients who have been wrongly treated for food poisoning will show a mass due to an appendiceal abscess. A scout film of the abdomen will usually show free air if perforation has occurred.

### Treatment

In a case of uncertainty no treatment should be given that might mask the true symptoms. This would exclude antibiotic therapy and the use of sedatives and antispasmodics. In any case of crampy abdominal pains cathartics and enemas must be avoided since they may cause a perforation or death. This fact must be constantly publicized in order to avoid disastrous self medication. Early operation is the only safe procedure even though a patient's symptoms may appear to be subsiding. This apparent subsidence often happens immediately after a perforation.

**WARNING** In any case of suspected acute appendicitis a careful general physical examination must never be overlooked. Other acute diseases may produce symptoms resembling those of appendicitis among them pneumonia at times coronary occlusion tubo ovarian disease and urinary tract disease. Any of these may also accompany appendicitis.

### CHRONIC APPENDICITIS

When Fitz in 1886 described acute appendicitis and later McBurney and Morris described their respective points of tenderness as indicating appendicitis and demonstrated how easy it was to remove an appendix there occurred an era of indiscriminate removal of appendices from all persons exhibiting such tenderness. When patients who had had this operation returned in droves to their surgeons with no relief of their preoperative symptoms and often with new and more serious symptoms a reappraisal was undertaken. Disregarding the fact that most of these operations had been done without any preoperative study to indicate whether such surgery could be expected to help and often with complete neglect of other lesions surgeons simply fell back on the assumption that there was no such disease as chronic appendicitis. This conception is still prevalent and it is often difficult to get a surgeon to remove an appendix which is definitely the cause of chronic often disabling symptoms.

adherent to the ureter. It is best brought out by films taken with a catheter in the ureter (Fig. 74 b). All these indirect findings should be looked for when a patient who has had an appendectomy has failed to be relieved of symptoms.

### Diagnosis

When a patient presents the symptoms mentioned above is tender over McBurney's and Morris points and shows a pathological appendix on x-ray study, it is reasonable to make a diagnosis of chronic appendicitis. This diagnosis alone is of little significance however from the standpoint of treatment. The important findings are the evidences of complications due to the diseased appendix and of coexisting diseases elsewhere as determined after complete study. No patient should be operated upon because of the appendiceal findings alone.

### Treatment

*Prophylaxis* is important. All focal infections including dead or pyorrheic teeth or retention cysts and infected tonsils should be removed.



Fig. 72



Fig. 73

Figure 72. X-ray film showing retrocecal appendix beaded (at arrow) cecum and ascending colon empty.

Figure 73. Ileocecal adhesions due to appendix (barium enema). 1 cecum, 2 ileum filled in position, 3 appendix adherent to posterior wall of ileum.

Cardiac and pulmonary lesions should be carefully ruled out. The abdomen may show signs more of a complicating lesion than of appendiceal disease. Tenderness in the right lower quadrant and especially at McBurney's and Morris points is suggestive. Pain at these points induced by pressure on either side of the abdomen, in the loin or in the rectum may suggest adhesions to neighboring organs. After repeated acute attacks a walled off abscess or noninfective obstruction with a mucocele may at times present a small palpable mass. When acute attacks occur or when ileal kinking causes obstruction the findings are those of an acute abdomen.

*Rectal examination* may locate tenderness high up especially to the right and at times a mass may be palpable. Proctoscopy will be of help in ruling out an intrinsic lesion of the rectum or sigmoid.

*Vaginal examination* may show right adnexal tenderness with difficulty in deciding whether it is due to an appendicitis or adnexal disease.

### X-ray Examination

*Direct Findings* Much has been made both of filling and lack of filling of the appendix as indicative of disease. It is certainly true that many apparently normal appendices allow barium to enter but it is equally true that the barium is forced out promptly. A residue in the appendix after the cecum has emptied has been generally agreed upon as evidence of a pathological appendix (Fig 72). It is entirely reasonable to suppose that it would occur when the wall of the appendix and its aperture are stiffened and held open in chronic appendicitis or some other cause of infiltration. If the chronic disease has resulted in obliteration or obstruction of the lumen the appendix will fail to fill with barium and will not be seen in the x-ray film. Feculiths and multiple strictures of the appendix are also demonstrable. Fixation of the appendix to the terminal ileum and cecum can be seen with a barium enema (Fig 73). The reason for the failure to recognize the validity of a diagnosis of chronic appendicitis from these findings is the fact that it was formerly held to be an indication for an appendectomy. The failure to help a patient by this operation based on such findings alone brought the diagnosis instead of the ill advised operation into disrepute.

*Indirect Findings* These may be *functional* those of upper gastrointestinal irritability or pylorospasm due to retrostalsis or of colonic irritability due to direct irritation from a diseased or adherent appendix. *Organic findings* to be looked for in a gastrointestinal x-ray study are cecal or ileal deformities and much more rarely gastric or colonic deformities due to adhesions (Fig 74 a) also the evidences of concomitant diseases such as ulcer, gallbladder disease and colonic lesions.

*Pycnography* done at the forty eight hour period after a barium meal showing an appendix residue will usually reveal when the appendix is

adherent to the ureter. It is best brought out by films taken with a catheter in the ureter (Fig 74 b). All these indirect findings should be looked for when a patient who has had an appendectomy has failed to be relieved of symptoms.

### Diagnosis

When a patient presents the symptoms mentioned above is tender over McBurney's and Morris points and shows a pathological appendix on x-ray study it is reasonable to make a diagnosis of chronic appendicitis. This diagnosis alone is of little significance however from the standpoint of treatment. The important findings are the evidences of complications due to the diseased appendix and of coexisting diseases elsewhere as determined after complete study. No patient should be operated upon because of the appendiceal findings alone.

### Treatment

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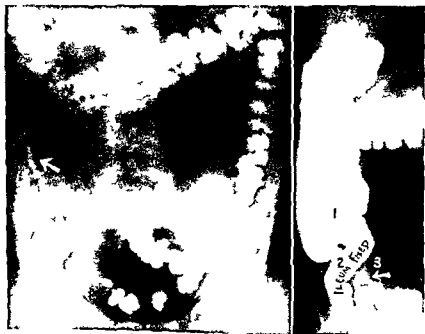


Fig 72

Fig 73

*Figure 72* X-ray film showing retrocecal appendix (labeled 1 at arrow); cecum and ascending colon empty.

*Figure 73* Ileocecal adhesions due to appendix (barium enema) 1 cecum 2 ileum fixed in position 3 appendix adherent to posterior wall of ileum.

a



b

Figure 74 a Beaded appendix and diverticula 1 Long beaded appendix tip in pelvis 2 diverticula ascending colon 3 diverticula descending colon and sigmoid Transverse colon (not in picture) also showed diverticula (Used through the courtesy of John B D Albora M D ) b Relation of appendix to ureter 1 Barium in appendix 2 cath xes in ureter

Treatment of any other infections such as sinus, genitourinary or rectal infections is of great importance. Many times all symptoms and findings of chronic appendicitis will disappear when such a program is carried out and acute episodes can be avoided.

**General Treatment** This includes attention to the patient's general condition, exercise, hygiene and particularly to the prescription of a well balanced diet (p. 21) with added vitamins and minerals. No particular restrictions to the diet are necessary; plenty of residue or "smoothage" helps to encourage cecal and therefore appendiceal function. No medication is desirable except for a short time. Heavy mineral oil, 1 tablespoonful at bedtime, may help to institute regular colonic function at the beginning.

**Surgical Treatment** No patient should be operated upon because of a diagnosis of chronic appendicitis alone. The results are usually not good. Operating because of persistent pain or disability supposed to be due to the appendix is also unsatisfactory.

**INDICATIONS FOR OPERATION** 1. Recurrent attacks of definite acute appendicitis are usually considered an indication to prevent future attacks.

2. Complications such as adhesions and kinks which have been definitely shown to be present in a patient who has failed to obtain relief from the measures recommended above require operation for relief. The surgeon, besides being fully acquainted with the preoperative findings, should make a careful general exploration. A Meckel's diverticulum is easily overlooked.

3. Appendectomy in the course of an operation for any abdominal or pelvic lesion when it will not add to the risk of the operation is advisable, especially if the preliminary study has disclosed evidence of a pathological appendix.

**POSTOPERATIVE PATHOLOGY** Removal of a diseased appendix will frequently not only fail to relieve many of its complications but may produce new ones. A small piece of the base of the appendix left in at operation may prove confusing upon subsequent x-ray examination. Invagination of the stump may cause a cecal defect and some narrowing of the cecum. Freed adhesions may re-form and new adhesions to neighboring organs may develop, producing symptoms and possibly providing a band for subsequent herniation. *Appendicitis rarely occurs alone*; it is usually associated with other gastrointestinal diseases, including particularly peptic ulcer, gallbladder disease and colitis, aside from the diseases considered etiological factors.

### Prognosis

Too many patients have had appendectomies performed for no good reason, often with resulting adhesions which further aggravate the symp-

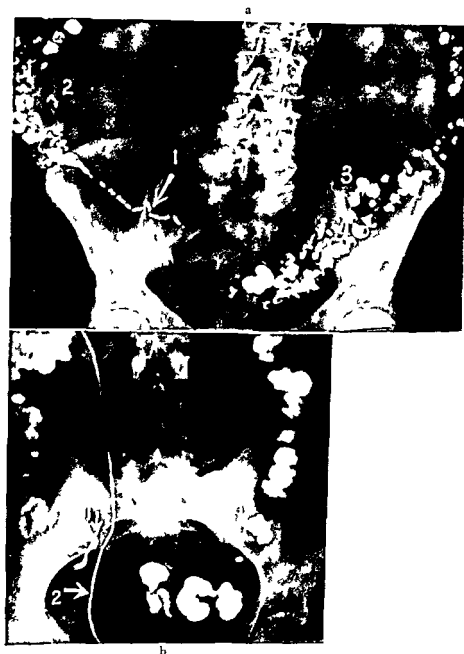


Figure 74 a Beaded appendix and diverticula 1 Long beaded appendix tip in pelvis 2 diverticula ascending colon 3 diverticula descending colon and sigmoid Transverse colon (not in picture) also showed diverticula (Used through the courtesy of J. m. B. D. Alborn, M.D.) b Relation of appendix to ureter 1 Barium in appendix 2 catheter in ureter

indirect trauma from external violence. A normal appendix is rarely affected by trauma although in the case of an adherent organ or one filled with fecaliths an acute attack may be instituted. Trauma severe enough to injure the appendix will invariably injure surrounding organs and the treatment will usually be surgical if the patient survives the injury.

## The Rectum and Anus

### General Discussion

Although the care of diseases of the rectum and anus has become a specialty generally recognized as a subspecialty of surgery, it is important for every clinician to know enough about this region to be able to recognize its diseases and to recommend proper treatment. There has been too much carelessness, too much actually harmful treatment in this field. It is usually dangerous when a patient complains of rectal distress or bleeding, to prescribe some hemorrhoidal suppository, an enema or a sitz bath and to give sedatives. Many patients with cancer treated in this way are found later with an advanced incurable lesion. Certain facts about the rectum must be emphasized (Fig. 75).

### ANATOMY

The rectum or pelvic colon itself begins at the rectosigmoid junction and ends at the internal sphincter, the extension below this between the internal and external sphincters being called the anal canal. It rests on the pelvic floor in contact with the other pelvic organs and measurement by means of a proctoscope discloses its length as averaging about 5<sup>1</sup>/<sub>2</sub> inches. The anal canal is 1.2 inches long. The rectum is comparatively narrow above, dilated at the middle, which is called the ampulla, and narrower again below. It is curved and fixed in position except at the rectosigmoid junction or angle where there is usually sufficient mobility to permit a proctoscope to be pushed through into the sigmoid. Like the colon, its wall has four coats: mucosa (with muscularis mucosae), submucosa and the muscular and serous coats. The mucosa is folded transversely or obliquely to form the crescentic rectal valves. <sup>1</sup> There are usually three or four in number, originating at the sides from the submucosa. The topmost valve is about 1<sup>1</sup>/<sub>2</sub> inches below the rectosigmoid junction. Longitudinally the mucosa at the lower end just above the sphincter is gathered into folds or pleats about 1<sup>1</sup>/<sub>2</sub> inch in length.



toms of the original real cause of them. Careful study and medical care will usually give excellent results even when a patient has shown definite evidence of a pathologic appendix. Operation should be performed only when one of the three indications previously mentioned is present in order to avoid persistent symptoms after operation.

### **NEOPLASMS OF THE APPENDIX**

Mycosis and polyps are rarely encountered. Endometriosis may involve the appendix (p. 395). Carcinoids now considered malignant are rarely found in removed appendices. Carcinoma may be primary in the appendix but is usually a part of a cecal carcinoma. Sarcoma has been found. Melanoma is a rare finding. None of the neoplasms can be recognized except at operation performed because they have caused obstruction thus producing symptoms of an acute appendicitis. At times a mucocele may be found.

### **General Diseases Affecting the Appendix**

General diseases may involve the appendix. Intestinal or peritoneal tuberculosis will often show tubercles in the appendix. Ileocecal tuberculosis in the acute stage is often mistaken for acute appendicitis. In typhoid fever the lymph follicles of the appendix may become involved with acute symptoms requiring operation. Lymphadenitis from any cause may also involve the appendix. Actinomycosis often affects the ileocecal region and appendix. Other mycotic diseases are discussed in the chapter on those diseases (p. 625). For parasitic diseases affecting the appendix see page 134.

### **Foreign Bodies in the Appendix**

Foreign bodies have long been associated in the lay mind as causes of appendicitis. Experimentally it has been shown that seed, BB shot and other foreign bodies introduced into the appendix are usually promptly expelled. If not, obstruction may result causing either appendicitis or mucocele. With the lumen so small it is probable that rarely do any foreign bodies pass into the appendix unless its cecal opening is held open by disease such as induration or adhesions. I have seen an acute appendicitis due to a tooth pick protruding from the tip of the appendix. Parasites have been known to enter the lumen. Opaque foreign bodies may rarely be seen by careful x-ray study. If they fail to be expelled, operation is indicated.

### **Trauma of the Appendix**

Injury to the appendix and its blood vessels as a result of trauma occurs rarely but must be considered in any case of violent injury. *Direct trauma* may result from bullet or stab wounds or from severe crushing injuries.

eight to ten in number. These are known as the columns of Morgani and between their lower ends or brises are small cup shaped folds called anal valves. Along the free edges of the valves are usually from two to six small tentlike projections called papillae. Behind each valve lies a pocket. These are called the *crypts of Morgani*. They normally secrete mucus which is squeezed out and smeared on the fecal mass by pressure as it passes through lubricating its surface. The lining of the *anal canal* shows a transition from mucosa to skin. At its distal end is the anus or anal orifice the skin of which is darker than the surrounding skin and is thrown into folds. It contains sweat and sebaceous glands and hair follicles. The *internal sphincter* is a ring of involuntary muscle. The muscles of the walls of the rectum are supplemented by the larger more powerful accessory muscles the most important the levator ani. The external sphincter controls the anal orifice.

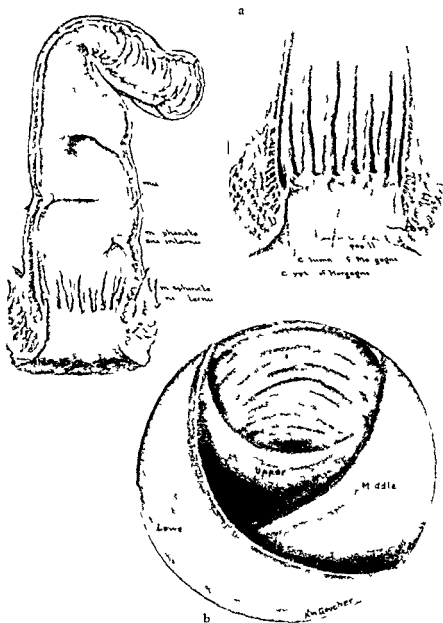
The *blood supply* is plentiful and there are many lymphatics. The *hemorrhoidal plexus* of veins is most important because deficient valves and pressure from the sphincter tend to produce enlargement resulting in hemorrhoids. The internal plexus lying above the anorectal line lies in the submucosa the external or inferior plexus lies outside the muscular layer. The internal plexus drains eventually into the portal vein the external plexus into the inferior vena cava.

The *nerve supply* is of importance. The rectum above the internal sphincter gets its nerve supply from the sympathetic and parasympathetic nervous systems and therefore is insensitive to pain. The anal canal below this gets its innervation from the cerebrospinal system so that this region is sensitive to pain increasingly toward the anus. Surrounding the rectum are various spaces or fossae the largest being the ischioanal fossa. The existence of these spaces together with the many glands crypts and papillae makes the rectum susceptible to infection. However the rectum which of course is always contaminated with millions of bacteria can stand a great amount of trauma even perforation without becoming infected or causing infection in contiguous tissues.

#### PHYSIOLOGY

The essential function of the rectum is the expulsion of the contents of the colon pushed into it from the sigmoid. This is aided by secretion of some mucus to act as a lubricant. There is also some absorption of fluid through the mucosa if the content is delayed in expulsion resulting in decreasing moisture in the feces up to the point of inspissation. This absorptive function is utilized in rectal feedings. Five or 6 ounces of water saline solution and nutrient if slowly introduced at body temperature will be absorbed promptly if the rectum contains no feces.

*The Feces* In addition to water the feces normally consist of indigestible and undigested material plus secretions of the upper intestine



**Figure 75** Normal rectum and anus *a* Longitudinal view of the anus rectum and lower sigmoid colon In the lower rectum are seen the columns of Morgagni the 3 rectal valves or valves of Houston are shown as are the rugae of the sigmoid Coronal section of lower rectum and anus showing anal valves valves or crypts of Morgagni (the columns of Morgagni somewhat accentuated) papillae external and internal sphincter muscles *b* Proctoscopic view showing the lower or inferior middle and upper or superior valves of Houston Above the upper valve the rugae of the sigmoid are discernible (Bacon Anus Rectum Sigmoid Colon 3rd ed J B Lippincott Company)

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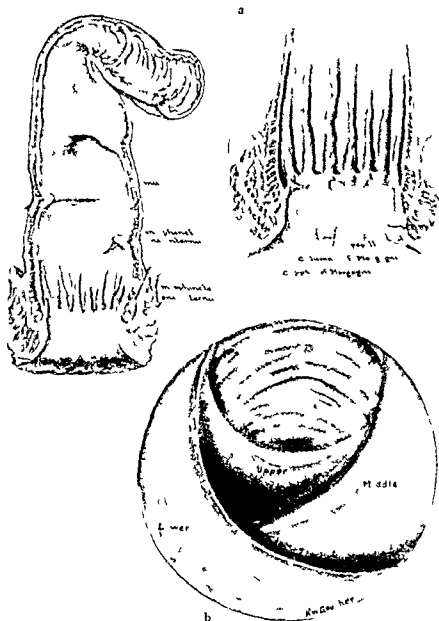
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tioned in the discussion of physiology irritation may result from *ingested irritating materials* such as chemicals foreign bodies—especially sharp ones—foods or other substances to which the patient is allergic and antibiotics. It may result from *rectally injected or inserted materials* such as chemicals foods medications enema tips rectal dilators or foreign bodies. It may also at times be due to contiguous tumors inflammations or foreign bodies such as pessaries in neighboring organs.

*Infection* may result from ingested bacteria from bacteria growing in the colon from infections in contiguous organs by direct extension from distant foci of infection by the hematogenous route or by direct infection from material deposited in the rectum.

*Infestation* usually occurs as a result of ingestion of parasites or ova but they may also be introduced directly through the anal orifice.

*Allergic reactions* may result from ingested allergens such as foods chemicals and drugs or from inhaled pollens smoke or dusts.

*Injury* may be caused by swallowed sharp articles such as pins nails and glass which have passed through the whole gastrointestinal tract with or without other injuries on the way down. More often it is caused by rectal instrumentation such as the use of proctoscopes bougies enema tips or operative procedures by the doctor or nurse by self introduced dilators or other foreign bodies for constipation or for perverted sexual gratification or by lacerations at parturition or their repair.

### SYMPTOMS

In general rectal disease should be suspected when a patient has a change in bowel habit diarrhea constipation or distress or pain caused or relieved by defecation. Symptoms obviously originating in the pelvis although referred in various directions suprapubically to either side or to the low back also should occasion a suspicion of some rectal condition either primary or secondary. Anal spasm pain protrusion or discharges of mucus pus or blood are obvious evidences of anal or rectal disease. Retrostaltic symptoms even when explainable on the basis of other diseases may result from anal and rectal irritation and should call for a thorough rectal study.

The symptoms caused by rectal diseases vary according to the nature of the disease its location and the functions of the part affected.

*Direct irritation* may cause increased motility and tonus with frequent peristalsis from above producing diarrhea. Milder irritation may cause only sphincter spasm and constipation.

*Infection* in addition to irritation will often cause pain which may radiate to the lower abdomen to either side or to the lower back. It will usually be accompanied by fever leukocytosis and other general evidences of infection and may spread to surrounding organs or to the portal system producing symptoms there. Infections of the mucosa

liver and pancreas debris from shedding of mucosal cells and millions of bacteria both dead and viable. The feces are stored in the sigmoid being held there as a result of a sphincter like action at the rectosigmoid junction and by the fact that rectal tone is higher than that of the sigmoid. A sudden more powerful peristaltic wave in the sigmoid usually following ingestion of food at varying intervals will overcome these obstructions and force feces into the rectum producing a sensation of fullness, rectal peristalsis and a desire to defecate. With the help of the accessory muscles of defecation, the abdominal muscles and the levator ani particularly and with relaxation of the sphincter, the fecal mass is expelled. The factors determining the frequency of defecation and the character of the contents have been discussed under the subjects of Constipation and Diarrhea (pp. 320-313).

So far as the rectum is concerned failure to respond to the "call of nature" when feces enter the rectum and the holding in of the feces by forcible contraction of the voluntary external sphincter results in their retention in the rectum. Not only does this cause dehydration of the feces but also the continued pressure and the sphincter contractions interfere with venous return tending to produce hemorrhoids. Likewise in diarrhea the continued contraction of these muscles in an effort to control or prevent expulsion has a similar effect on the veins. Diarrheal stools usually loaded with bacteria and various irritating secretions and admixtures cause irritation of the anal and rectal mucosa tending to produce inflammation, ulceration and bleeding. Such pathological changes occurring in the upper rectum may cause no direct symptoms except a fullness or desire to defecate together with retrostaltic symptoms. In the anus and in the anal canal they may cause severe pains producing not only retrostaltic symptoms up to the point of vomiting but also often psychological effects so that patients with such symptoms may be treated as neurotics or psychotics if the cause has not been determined. This indicates the need for thorough rectal examination of every patient with otherwise unexplainable retrostaltic or psychiatric symptoms before resorting to abdominal exploratory operation or elaborate psychiatric care.

#### PATHOLOGY

The various pathological conditions of the colon mentioned on page 309 are also found in the rectum modified by the structure and function of this organ. The pathology will be discussed under the description of each disease.

#### ETIOLOGY

Diseases of the rectum and anal region may be caused by a variety of factors.

*Irritation* is an important factor. In addition to the causes just men-

cervix or uterus or an infected or otherwise diseased bladder or prostate anteriorly, an inflamed appendix or ureter on one side or a psoas abscess posteriorly. The pressure of the finger against the mucosal glands or a fistulous tract may express pus, blood or mucus and these should be watched for and examined. The size, consistency and conformation of the prostate, cervix and uterus can easily be determined. Rectal neoplasms can be felt and it has been estimated that more than half of all rectal lesions are within reach of the examining finger. A prolapsed or intussuscepted bowel will may be felt and is difficult to differentiate except by proctoscopy. Metastatic cancer implanted in the cul de sac, the hard irregular Krukenberg tumor can be felt when the finger is pushed far up.

### INSTRUMENTAL EXAMINATIONS

All instrumental examinations are preferably made with the instrument well lubricated with a water soluble clear lubricant. The most useful instruments are shown in Figure 76.

#### Speculum Examination

Speculum examination with a branched instrument like a vaginal speculum is the best way to examine the anal orifice and the anal canal. When dilated and gently rotated the speculum gives a good view of hemorrhoids, fissures, ulcers, neoplasms and inflammatory reactions in the anal canal and lower rectum. By dilating the sphincters it prepares the rectum for examination with the rigid endoscopes. In-

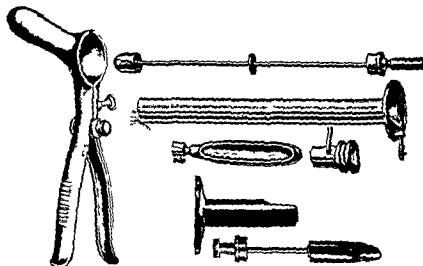


Figure 76 Pratt rectal speculum ano-cope Tuttle proctosigmoidoscope



starting in the wall and extending through the mucosa will usually be demonstrable by cultures of the material discharged from the rectum

*Infestations* will produce irritation may be accompanied by bleeding and may show an eosinophilia. Larger worms will be seen in the stools and may be described accurately by the patient

*Allergic reactions* must be suspected when other allergic manifestations have been present when onset is sudden when careful history suggests certain foods or drugs as possible causes or when attacks are seasonal in occurrence. Allergic manifestations may produce symptoms easily mistaken for any other rectal diseases. The whole subject is discussed in the chapter on Allergy (p. 87)

### PHYSICAL EXAMINATION

As in any gastrointestinal disease a complete physical examination is always essential. Search for focal infections for chest cardiac genitourinary spinal or other diseases must be complete and thorough. The abdomen must be examined for tenderness muscle guarding spasm or rigidity tumors and palpable or enlarged organs. Hernias enlarged glands and reflexes should be checked. The condition of the skin may be important.

Examination of the rectum is accomplished by inspection and by digital and instrumental examinations.

*Inspection* of the anal region aided by spreading apart of the buttocks and the anal orifice will disclose protrusions fistulous openings ulcerations discoloration of the skin and anal discharges of mucus pus or blood.

*Digital examination* is best accomplished with the patient in the left lateral or Sims position. It is usually made with a gloved finger or one protected by a finger cot. If after putting on the cot the finger is thrust through a double layer of toilet tissue or a square of absorbent cotton the examination can be accomplished without soiling the fingers or hand. Albu of Berlin demonstrated this to me in 1909 saying that after such an examination you could sit down to dinner without washing your hand. The finger well lubricated should be inserted slowly to avoid pain and spasm. If a painful anal lesion such as an ulcer or distended hemorrhoid is encountered an analgesic ointment should be applied and allowed to relieve the spasm and pain before the finger lubricated with the same ointment is again introduced. In the anal canal protrusions from hemorrhoids enlarged papillae or neoplasms depressed areas or a roughness suggesting induration may be felt. Passing through the internal sphincter which when relaxed suggests a spinal lesion the finger tip will no longer cause pain unless it impinges on an inflamed or distended lesion which is causing perirectal or peritoneal stretching. This would include an infected or enlarged

the examination is being made. In cases of bloody diarrhea it is best to examine without preparation first so that not only can the lesions be seen unaffected by treatment but also so that cultures and smear will be more reliable. To be sure about neoplasms or other definite lesions the rectum should be empty and clean. This is best accomplished by giving 1½ ounces of castor oil the night before the examination and making the examination toward noon or a little later the next day. Enemas never produce as good a preparation although the new type of enema performed by injecting a cleansing solution from a collapsible plastic bag is very good.

Proctoscopy should not be performed except under guidance of an expert until the examiner has had considerable experience. Frequently a tyro will perforate the rectal wall because of inexperience. An experienced examiner never hesitates to stop the examination when he cannot see the lumen or when feces obstruct it.

Cultures of contents of rectum and sigmoid obtained by smear or suction are of great value.

*Biopsy* by means of a special instrument can usually be performed easily and safely through the anoscope or proctoscope. Care must be used not to perforate the wall. This is the surest way to make a diagnosis of neoplasms, inflammatory exudates, specific infections and schistosomiasis.

#### X RAY EXAMINATION

Because the shadow of a barium filled rectum is greatly obscured by the shadows of the pelvic bones and lower spine x ray study is not as satisfactory as in other parts of the gastrointestinal tract.

*Direct study* of the rectum by fluoroscopy during administration of an opaque enema may at times show defects or narrowings in the rectum or sigmoid even when there is no obstruction to the flow of



Figure 77. Knee-chest position. Chest on table. Thighs vertical. Knees slightly separated. (Bacon: Anus, Rectum, Sigmoid Colon, 3rd ed. J. B. Lippincott Company.)

dentially it is useful for dilating the sphincters to relieve pain and spasm due to fissures or other lesions

### Anoscopy

The anoscope is really a short proctoscope usually 2 to 3 inches long and often with an opening on one side to give a wider view. It is not as useful as a speculum for inspecting the anal orifice but is better for the anal canal especially upon withdrawal when the mucosa follows it and bulges into the distal end giving a good view of the stretched mucosa or the descending mucosa of a rectal or sigmoid prolapse.

### Proctoscopy

The proctoscope is usually from 7 to 12 inches long and its diameter does not need to be as small as is seen in many instruments. A  $\frac{3}{4}$  or 1 inch diameter is usually entirely satisfactory even in a 10 or 12 inch proctoscope. As proctoscopy should include inspection not only of the rectum but also of as much of the sigmoid as possible the terms proctosigmoidoscope and "sigmoidoscopy" are really superfluous. I prefer to speak of proctoscopy even when the 12 inch instrument has been inserted well into the sigmoid. The methods of illumination whether distal or proximal are not important. Their choice is a matter of individual preference.

*Proctoscopy* is best performed in the knee chest position (Fig 77) or on one of the tables made for this examination. The essential feature is that the thighs should be exactly vertical the body bent acutely forward allowing the force of gravity to cause the colon to fall into the upper abdomen pulling the sigmoid upward and straightening the rectosigmoid angle. As soon as the proctoscope has passed the internal sphincter the obturator should be removed and further advance should be guided visually. Never should the tip be advanced except when the lumen thereof can be seen. Gently swinging the tip from side to side will often disclose the lumen. Inflation of the rectum by closing the proximal end with a window and inflating air with a bulb will often open up a lumen but there is grave danger of perforating the bowel. Inflation is therefore to be condemned even in the hands of an expert.

Proctoscopy should not be performed in a badly debilitated patient in the presence of cardiac or pulmonary conditions associated with severe dyspnea in the presence of an abscess which might be ruptured by the examination or when pain is so great that the patient cannot cooperate by keeping quiet and breathing gently through the mouth.

*Preparation* for the examination depends on the condition for which

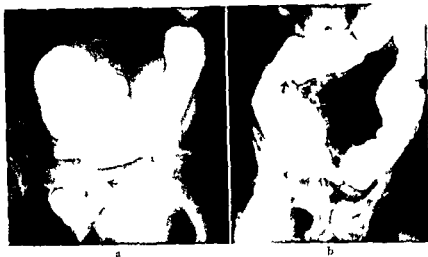


Figure 78 a Cancer of rectum. Note irregular outline at arrow due to cancer. Rectum filled above and below. b Benign stricture of rectum (from pelvic adhesions). Note smooth outline of narrowed rectum. Colon dilated above especially cecum and ascending colon due to pyloric Cannon's ring (arrow).



Figure 79 Cancer of rectosigmoid (2 studies on one film) 1 72 hour accumulation of barium behind spastic Cannon's ring shown at arrow 2 3 Rectum filled with barium enema up to obstructing cancer shown at arrow 4

barium mixture Defects due to external pressure from the uterus or adnexa the bladder prostate or retroperitoneal neoplasms may also be detected Films taken immediately after the rectum and sigmoid have been filled may bring out these defects and if taken after expulsion may show ulcers filled with the barium or some barium backing up behind a partial obstruction Defects due to foreign bodies of various kinds and protrusions due to diverticula or fistulas may also be seen Usually the study of the rectum is a part of a general barium enema study and lesions may easily be overlooked which can be seen easily through the proctoscope.

*Indirect evidence* of a rectal or lower sigmoid obstruction may be seen in a scout film Such obstruction in its early stages usually causes more or less spasm of the sphincter known as *Cannon's ring* at the junction of the first and middle thirds of the transverse colon This results in dilatation of the colon proximal to this point which can be seen distended with gas Later when the sphincter is relaxed the whole colon becomes distended to the point of obstruction When an obstruction is suspected barium should not be given by mouth If it has been given the ascending colon will be filled with opaque substance On the other hand it has been noted that the finding of a dilated rectum is often an accompaniment of an obstruction of the lower sigmoid The combination of a dilated proximal colon and rectum is therefore suggestive of lower sigmoid obstruction

#### LABORATORY EXAMINATIONS

Examination of feces and of rectal discharges or aspirates includes of course the routine tests for food remnants for evidences of insufficient digestion of meat fibers starches and fats and for admixtures such as blood mucus pus parasites and ova Specifically of help in rectal diagnosis are cultures for bacteria Smears and scrapings should be studied for pinworms and their ova motile amebae and their cysts and particularly for cytologic study by the Papanicolaou method

Other examinations such as blood cell counts sedimentation rate chemistries and serology should be done as a routine Gastric analysis sputum examinations urinary studies and vaginal smears may give valuable additional information

#### TREATMENT

##### Surgical Treatment

The feeling that all treatments for rectal disease belong in the realm of surgery is distinctly not true It is true that a number of rectal conditions can be treated only by surgery These include of course the removal of neoplasms benign and malignant the incision of abscesses the excision of cysts fistulas and deep fissures the repair of prolapses

safe to pass these except by direct visual guidance through an anoscope or proctoscope. The endoscope is passed to the stricture and the tip of a dilator the size of the opening well lubricated is gently pushed through. If not much resistance is offered the next size may be tried but too much force must be avoided in order to prevent perforation of the wall. The dilatation causes moderate pain and may cause bleeding. It should not be repeated more than once or twice a week, the rectum meanwhile being soothed by oil or gelatin enemas. Dilatation to a  $\frac{3}{4}$  inch diameter is usually safe and sufficient. As a rule the dilatations will need to be repeated at lengthening intervals finally once or twice a year even though causing no symptoms. Sudden agonizing pain should create a suspicion of perforation and the patient should immediately be hospitalized and treated as described under Trauma (p. 456). I have done successful dilatations in patients with strictures from operations, injuries, ulceration and x-ray and radium burns.

### Anomalies and Displacements of the Rectum

#### CONGENITAL AND DEVELOPMENTAL DEFECTS

Congenital and developmental defects are extremely rare and are usually discovered at birth or soon afterward. They generally consist in



Figure 80 a Wales bougie used as dilator b Bougie in rectum

and severe injuries and the correction of anomalies. After operation incontinence is a distressing symptom and may require further operation. Other diseases such as ordinary fissures, hemorrhoids, infections, pruritus, injuries and foreign bodies may often be treated successfully without surgery or may be benefited by medical care preliminary to major or minor surgery. A discussion of surgical operations and the forms of anesthesia suitable for each kind does not come within the purview of this book. There are many textbooks on proctology which cover this field comprehensively.

### Medical Treatment

In general medical treatment consists in prophylaxis, dietetic care, medicinal, endocrine, hormone and immune therapy, and the use of laxatives, enemas and suppositories. These are discussed in the introductory chapter (p. 17) as are radiotherapy and electrodesiccation (p. 451). Psychotherapy, often necessary in the presence of chronic rectal symptoms, is discussed in the chapter on Psychosomatic Disturbances (p. 131).

The indications for the use of these various treatments will be discussed in considering each disease.

### COMPLICATIONS

In general the complications of rectal diseases consist in spread of the disease to neighboring organs or upward into the colon, perforation of the rectal wall either spontaneously as a result of disease or induced by rough handling or instrumentation, or the dissemination of disease from a rectal inflammatory or malignant focus through blood or lymph channels.

*Complications following surgery* even minor anorectal surgery may occur immediately after operation or later. *Immediate complications* include fecal impaction, hemorrhage from the wound, infection and excessive pain. By delaying the healing process any of these complications may cause persistent poor function. *Later complications* include stenosis, formation of fissures, fistulas, tags, excessive granulation, pseudoadenomas, papillitis or cryptitis with resulting anorectal deformities and spasm or excessive relaxation resulting in incontinence. Careful operative procedures, frequent inspection and digital examination after operation and attention to diet both before and afterwards will prevent these complications. Operation may be required to correct them.

*Incomplete stenosis* may at times be treated successfully by means of gradually increasing dilatation. Mechanical dilatation by means of branched dilators is too risky to be recommended. The safest dilators are bougies made of solid flexible rubber about 12 inches long with gradually tapering tip (Fig. 80). They come in various sizes. It is not

safe to pass these except by direct visual guidance through an anoscope or proctoscope. The endoscope is passed to the stricture and the tip of a dilator the size of the opening well lubricated is gently pushed through. If not much resistance is offered the next size may be tried but too much force must be avoided in order to prevent perforation of the wall. The dilatation causes moderate pain and may cause bleeding. It should not be repeated more than once or twice a week, the rectum meanwhile being soothed by oil or gelatin enemas. Dilatation to a  $\frac{3}{4}$  inch diameter is usually safe and sufficient. As a rule the dilatations will need to be repeated at lengthening intervals finally once or twice a year even though causing no symptoms. Sudden agonizing pain should create a suspicion of perforation and the patient should immediately be hospitalized and treated as described under Trauma (p. 456). I have done successful dilatations in patients with strictures from operations, injuries, ulceration and x-ray and radium burns.

### Anomalies and Displacements of the Rectum

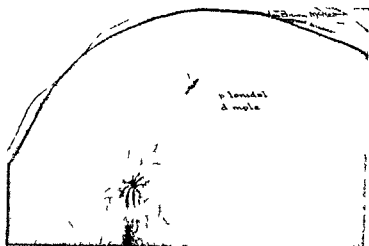
#### CONGENITAL AND DEVELOPMENTAL DEFECTS

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Figure 80 a Wales bougie used as dilator b Bougie in rectum





*Figure 81* Opening of a pilonidal sinus in the midline of the sacrococcygeal region. Note slight dimpling of skin and the protrusion of a few hairs (Bacon: Anus Rectum Sigmoid Colon 3rd ed J B Lippincott Company)

absence of anus or rectum or both; more or less occlusion of the anus by membranes or fibrous tissue or arrested descent of the rectum. All are associated with absence of meconium or the passage of very little at a time with evidences of intestinal obstruction. Abnormal location of the anus at some point in the perineal, scrotal or sacral region and opening of the rectum into a neighboring organ such as the bladder, urethra, vagina or uterus are associated with the discharge of meconium from these regions. All these anomalies require operation as soon as detected. Congenital megacolon or Hirschsprung's disease in which there is supposed to be an absence of the myenteric ganglia causing absence of peristalsis in the rectum and sigmoid with marked colonic dilatation above is discussed on page 333.

### PILONIDAL SINUS AND CYST

Pilonidal sinus is a congenital defect lying in the midline in the sacrococcygeal region. It consists in an invagination of the skin like a dimple forming a tract in which are collected hairs, shed skin and secretions resulting in a cyst. If infected it tends to burrow and becomes an abscess which may produce fistulous tracts. It may remain merely a dimple or may discharge some of its contents. Encountered at all ages it is most common in the second and third decades of life and is three times as common in men as in women. It is rarely seen in the colored race.

### Symptoms

There are usually no symptoms unless trauma or infection super-

venes Trauma results from prolonged sitting especially on a jolting object such as a horse or wagon It was called "cisson disease" during the war There is then pain on sitting or lying and staining of the clothes with the secretion occasionally bloody

### Diagnosis

The sinus can be demonstrated by inserting a probe into the "dimple" its depth can be determined by x ray study after injection of an opaque medium Occasionally a few hairs may project from the dimple and there may be surrounding redness Differentiation from isorectal fistula furuncle carbuncle osteomyelitis and actinomycosis must be made

### Treatment

Symptom free dimples require no treatment except cleanliness All others are best handled by a proctologist In some cases sclerosing agents may be useful in others operation is indicated All kinds of operative procedures have been recommended In general the more radical the procedure the better the result Recurrences are common when excision and immediate suture is done but unusual with open packing

### PROLAPSE AND PROCIDENTIA

Prolapse is a general term used to describe abnormal descent of rectal mucosa To avoid confusion it is best to consider prolapse only as a downward displacement of the sigmoid rectal or anal mucosa alone and procidentia as a downward displacement of all coats Both are in the nature of an intussusception Both are considered internal if protrusion is only in the lumen external if it extends beyond the anal orifice In all types there is a tendency for the prolapsed mucosa or wall to move upward and downward alternating the occurrence of the protrusion with its reduction This reduction may be accomplished spontaneously with or without elevation of the hips or lying on the left side by manual or instrumental pressure or by enemas When an external protrusion becomes permanent and irreducible it may require operation

### Etiology

In general prolapse may be due to immaturity of the pelvic organs in small children or to general debility emaciation or lack of muscle tone in the aged Any condition causing relaxation or paralysis of the sphincters or the levator ani may play a part Prolonged sitting in an erect or other unnatural posture at stool also predisposes to prolapse Specific mechanical causes of prolapse include hemorrhoids intrinsic rectal tumors inflammation and edema of the rectal wall hypertrophied

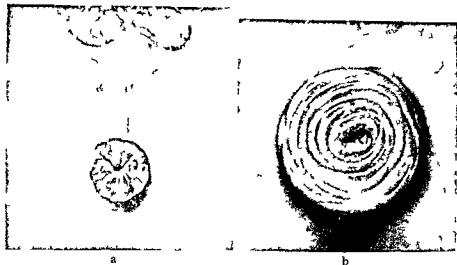


Figure 82 a Prolapse of the rectum The radial striations or furrows radiating from the center of the anal aperture can be seen b Procidentia showing striations circularly arranged (Bacon Anus Rectum Sigmoid Colon 3rd ed J B Lippincott Company)

papillae and hard stools partly because they cause increased straining and congestion External causes include pelvic and abdominal tumors weakness of the pelvic floor from injury or parturition damage direct injuries to the rectum by careless divulsion or instrumentation foreign bodies parasites and sodomy Any condition causing constant or occasional increased intra abdominal pressure will produce or aggravate the prolapse These would include straining at defecation or urination vomiting coughing sneezing horseback riding and tight constriction of the abdominal wall

### Symptoms

In internal prolapse there may be few if any direct symptoms Constipation is usually complained of often described as a sudden stoppage during the act of defecation sometimes followed by passage of mucus or a little blood After defecation there may be a feeling of something left behind At times there is a pain or an aching in the pelvic region lower back or down the thighs

The external protrusion is usually mistaken for hemorrhoids and may of course be initiated by hemorrhoids pulling down mucosa with them when they protrude At first the prolapse tends to be reduced spontaneously later it may require manual replacement after defecation It may occur at long intervals at first usually after a situation causing increased abdominal pressure and later may occur with increasing frequency until it becomes irreducible There is usually a bearing-down feeling or actual pain or full feeling in the pelvis lower backache and aching in the legs A mucous discharge occasionally blood stained may

occur with or independent of defecation. The stools may be hard and later may become diarrheal.

### Diagnosis

Inspection will show the external protrusion small or large. An anal prolapse shows a congested and edematous anal and perianal skin. A rectal prolapse will show longitudinal striations radiating from the anal orifice as a center. A rectal procidentia will present an oval firm protruding mass which may be from 2 inches to a foot in length. The surface shows circumferential folds of mucosa coated with glistening mucus and occasionally eroded or ulcerated. The whole mass may become congested and swollen. A finger can usually be inserted between the mass and the rectal wall.

The internal prolapse or procidentia may be within reach of the finger or may be seen through a proctoscope either of which may be passed between it and the rectal wall. The procidentia is of a firm consistency the prolapse is soft and may be missed. Either may be reduced when air enters the rectum upon insertion of the proctoscope and will then be missed. With the patient straining or bearing down the protrusion will usually recur so that it can be observed. Careful search must be made for possible causes such as those mentioned above. A cancer felt by the fingers or seen through the proctoscope may suddenly disappear when a prolapse is reduced.

### Complications

A persistent prolapse may show increasing ulceration of the mucosa or rarely may become strangulated or may perforate. The annoyance and discomfort and the interference with defecation usually compel patients to seek relief before these complications develop.

### Treatment

The removal or treatment of the causes is essential. This would include attention to hemorrhoids, removal of tumors and papillae and other treatments as indicated. Careful attention to diet, water intake and general hygiene is important. The diet should be well balanced and should provide sufficient soft bulk to encourage normal defecation (see Anticonstipation Diet p. 323). A tablespoonful of mineral oil at bedtime will provide sufficient lubrication from above. A retention enema of 5 or 6 ounces of warm oil at bedtime will add to the lubrication and may be used for a time. A small (6 to 8 ounces) cold water enema in the morning may be a help. A "baby syringe" can be used for this. A "hemorrhoidal suppository" at bedtime is a good substitute.

The protruding bowel must be pushed upward gently after defecation and any other time it recurs. It is best accomplished with the patient

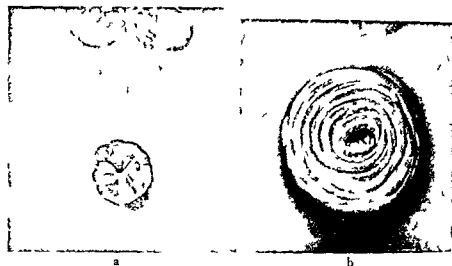


Figure 82 a Prolapse of the rectum. The radial striations or furrows radiate from the center of the anal aperture can be seen. b Procidentia showing striations circularly arranged. (Bacon: Anus Rectum Sigmoid Colon 3rd ed. J. B. Lippincott Company.)

papillae and hard stools partly because they cause increased straining and congestion. External causes include pelvic and abdominal tumors, weakness of the pelvic floor from injury or parturition, damage, direct injuries to the rectum by careless division or instrumentation, foreign bodies, parasites and sodomy. Any condition causing constant or occasional increased intra-abdominal pressure will produce or aggravate the prolapse. These would include straining at defecation or urination, vomiting, coughing, sneezing, horseback riding and tight constriction of the abdominal wall.

### Symptoms

In internal prolapse there may be few if any direct symptoms. Constipation is usually complained of, often described as a sudden stoppage during the act of defecation, sometimes followed by passage of mucus or a little blood. After defecation there may be a feeling of something left behind. At times there is a pain or an aching in the pelvic region, lower back or down the thighs.

The external protrusion is usually mistaken for hemorrhoids and may of course be initiated by hemorrhoids pulling down mucosa with them when they protrude. At first the prolapse tends to be reduced spontaneously, later it may require manual replacement after defecation. It may occur at long intervals at first, usually after a situation causing increased abdominal pressure, and later may occur with increasing frequency until it becomes irreducible. There is usually a bearing-down feeling or actual pain or full feeling in the pelvis, lower backache and aching in the legs. A mucous discharge, occasionally blood-stained, may

roids Protruding from the anus with defecation and often with some mucosal prolapse they may usually be easily pushed upward above the sphincter If protruding too long they may become inflamed and painful Ordinarily they may cause an itching or crawling sensation rarely a discharge Sudden hemorrhage into a papilla may cause severe rectal pain and is often mistaken for a thrombosis of a hemorrhoid They may be seen by spreading apart the anal folds or through a speculum and can be easily excised under local anesthesia The only danger is from infection or hemorrhage

*Cryptitis* is an inflammation of the crypts of Morgagni which are located a little above the papillae and the semilunar valves Cryptitis is considered a possible focus of infection causing distant lesions It has been considered a cause of pruritus ani of anal fissure and fistula and even of hemorrhoids Aside from causing at times severe rectal pains and sphincter spasm especially with defecation it may be accompanied by headaches insomnia nervous symptoms and various muscular aches and pains Retrostaltic gastrointestinal symptoms may also be present The inflamed crypts may be recognized by tenderness just above the anorectal line and may be seen through a speculum or anoscope by pulling down the semilunar folds with a hook Infection may cause abscesses followed by fistulas In such cases operation is of course indicated In uncomplicated cases oil enemas and hemorrhoidal suppositories often tend to relieve symptoms and may aid in clearing up the condition entirely In cases caused by allergy there is prompt subsidence when the offending allergen is avoided (See Gastrointestinal Allergy p 87)

#### ABSCESSSES

Abscesses of the anorectal region are common and it is essential that their early care be thorough Too many patients present themselves with fistulas which result directly from inadequate care of abscesses The fossae with a large amount of loose areolar tissue in this region the frequent irritation of the area by feces toilet paper enema tips enemas the fingers chafing from rough clothing the exposure to trauma the frequency of pelvic diseases impinging on this region and the constant presence of bacteria—all contribute to the development of these abscesses Debilitating diseases in general cause lowered resistance to infection Abscesses are to be looked for particularly in patients with diabetes tuberculosis and ulcerative colitis

The bacteria found in the abscesses may be the organisms resident in the colon streptococci and staphylococci and occasionally proteus and tubercle organisms The abscess caused by this infection may grow in size slowly or rapidly its margins constantly pushing into the surrounding loose tissues By the time it will open spontaneously

lying on the left side Gentle pressure with or without a lubricant will reduce most prolapses that are not too large It is necessary to see that the bowel is pushed above the internal sphincter Sometimes it is advantageous to press a pad against the anus afterward and try to keep it there Bed rest may be necessary for a short time By not permitting the protrusion to persist it will usually decrease in size and may finally not recur A prolapse which at first may seem too large to be reduced usually because of congestion and edema may be decreased in size by the application of compresses or mild astringents such as 0.5 per cent zinc sulfate or 2 per cent lead acetate Ulceration may call for application of silver nitrate Large irreducible protrusions require surgical care This may consist in various injections or one of the many operations which have been used by different surgeons

### Prognosis

Mild cases usually do well with the treatment as outlined After removal of hemorrhoids or other lesions there may be a tendency to some anal protrusion with straining at defecation but this can usually be handled easily After the more extensive operations recurrences are common

## Inflammatory Diseases of the Rectum

### PROCTITIS

The rectum is usually involved in any general inflammatory condition of the colon The various forms of colitis discussed in the previous chapter should be consulted in evaluating the diagnosis and treatment At times only the rectum or sigmoid may be involved in ulcerative colitis but the treatment would be the same Antibiotics are usually indicated in the rectal form but may in themselves cause trouble For the rectal involvement as seen through the proctoscope soothing or astringent enemas or suppositories or insufflation of astringent powders may be of some value Hot gelatin enemas may help to control bleeding (p 47) In amebic proctocolitis instillation of 5 or 6 ounces of an amebicide solution is to be recommended (See chapter on Parasites p 139)

### PAPILLITIS AND CRYPTITIS

These diseases occur with a generalized proctitis but may also occur usually together without such general involvement

The papillae from two to six in number lie along the anorectal margin and are triangular teatlike projections of mucosa When irritated by rectal inflammatory conditions or by traction from prolapsed hemorrhoids hard or frequent diarrheal stools or foreign bodies they hypertrophy occasionally growing so large as to be mistaken for hemor-

roids. Protruding from the anus with defecation and often with some mucosal prolapse they may usually be easily pushed upward above the sphincter. If protruding too long they may become inflamed and painful. Ordinarily they may cause an itching or crawling sensation rarely a discharge. Sudden hemorrhage into a papilla may cause severe rectal pain and is often mistaken for a thrombosis of a hemorrhoid. They may be seen by spreading apart the anal folds or through a speculum and can be easily excised under local anesthesia. The only danger is from infection or hemorrhage.

*Cryptitis* is an inflammation of the crypts of Morgagni which are located a little above the papillae and the semilunar valves. Cryptitis is considered a possible focus of infection causing distant lesions. It has been considered a cause of pruritus ani, of anal fissure and fistula and even of hemorrhoids. Aside from causing at times severe rectal pains and sphincter spasm especially with defecation it may be accompanied by headaches, insomnia, nervous symptoms and various muscular aches and pains. Retrostaltic gastrointestinal symptoms may also be present. The inflamed crypts may be recognized by tenderness just above the anorectal line and may be seen through a speculum or anoscope by pulling down the semilunar folds with a hook. Infection may cause abscesses followed by fistulas. In such cases operation is of course indicated. In uncomplicated cases oil enemata and hemorrhoidal suppositories often tend to relieve symptoms and may aid in clearing up the condition entirely. In cases caused by allergy there is prompt subsidence when the offending allergen is avoided. (See Gastrointestinal Allergy, p. 87.)

#### ABSCESSSES

Abscesses of the anorectal region are common and it is essential that their early care be thorough. Too many patients present themselves with fistulas which result directly from inadequate care of abscesses. The fossae with a large amount of loose areolar tissue in this region, the frequent irritation of the area by feces, toilet paper, enema tips, the fingers chafing from rough clothing, the exposure to trauma, the frequency of pelvic diseases impinging on this region and the constant presence of bacteria—all contribute to the development of these abscesses. Debilitating diseases in general cause lowered resistance to infection. Abscesses are to be looked for particularly in patients with diabetes, tuberculosis and ulcerative colitis.

The bacteria found in the abscesses may be the organisms resident in the colon: streptococci and staphylococci and occasionally pyocyanus, proteus and tubercle organisms. The abscess caused by this infection may grow in size slowly or rapidly, its margins constantly pushing into the surrounding loose tissues. By the time it will open spontaneously



lying on the left side Gentle pressure with or without a lubricant will reduce most prolapses that are not too large It is necessary to see that the bowel is pushed above the internal sphincter Sometimes it is advantageous to press a pad against the anus afterward and try to keep it there Bed rest may be necessary for a short time By not permitting the protrusion to persist it will usually decrease in size and may finally not recur A prolapse which at first may seem too large to be reduced usually because of congestion and edema may be decreased in size by the application of compresses or mild astringents such as 0.5 per cent zinc sulfate or 2 per cent lead acetate Ulceration may call for application of silver nitrate Large irreducible protrusions require surgical care This may consist in various injections or one of the many operations which have been used by different surgeons

### Prognosis

Mild cases usually do well with the treatment as outlined After removal of hemorrhoids or other lesions there may be a tendency to some anal protrusion with straining at defecation but this can usually be handled easily After the more extensive operations recurrences are common

## Inflammatory Diseases of the Rectum

### PROCTITIS

The rectum is usually involved in any general inflammatory condition of the colon The various forms of colitis discussed in the previous chapter should be consulted in evaluating the diagnosis and treatment At times only the rectum or sigmoid may be involved in ulcerative colitis but the treatment would be the same Antibiotics are usually indicated in the rectal form but may in themselves cause trouble For the rectal involvement as seen through the proctoscope soothing or astringent enemas or suppositories or insufflation of astringent powders may be of some value Hot gelatin enemas may help to control bleeding (p 47) In amebic proctocolitis instillation of 5 or 6 ounces of an amebicide solution is to be recommended (See chapter on Parasites p 139)

### PAPILLITIS AND CRYPTITIS

These diseases occur with a generalized proctitis but may also occur usually together without such general involvement

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defined with an elastic smooth base. As it becomes more chronic it may extend into the subcutaneous tissue even into the muscle becoming indurated and occasionally undermined as a result of infection. It is often accompanied by pruritus ani. The associated severe sphincter spasm causes it to be extremely painful. The pain at first occurs mostly during defecation later becoming constant and extremely debilitating. The pain may radiate into the bladder and genitals upward into the rectum and back and downward into the legs. Retrostaltic symptoms are at times so pronounced that an upper gastrointestinal lesion may be suspected. Women who are too bashful to mention anal symptoms for fear of local examination have at times been subjected to exploratory laparotomy. Insomnia due to pain, general irritability and neurosis are common accompaniments.

### Etiology

The causes of the ulceration are numerous.

1 The puckering and sphincter tone of the anal orifice favor maceration due to moisture.

2 Trauma is the most frequent immediate cause and may be due to (a) trauma or abrasion caused by careless examination instrumentation or treatment (b) foreign body such as an enema tip swallowed small bones or objects introduced for sexual gratification (c) hard constipated stools.

3 Excessive sphincter spasm as a result of straining at stool either from constipation or diarrhea or from a neoplasm local or higher up.

4 The ulceration may be part of a general disease process in the rectum such as syphilis, chancroid, lymphopathia venereum, gonorrhea, tuberculosis or ulcerative colitis or to a local disease such as proctitis, papillitis or cystitis.

5 Allergy is an important factor especially in the type of ulcers which occur at intervals (p. 86) are very painful for a few days heal without scarring and recur later. Complications include hemorrhoids, cryptitis and occasionally fistula in ano.

### Diagnosis

The pain as described above is characteristic. At times a large caliber stool will stretch the sphincter sufficiently to prevent spasm and will give relief from pain for a varying time. Inspection of the anal orifice by spreading apart of the buttocks may disclose the fissure or at least the "sentinal pile." Digital examination will encounter the spasm and in old cases the induration may be felt. The use of an analgesic ointment for lubrication helps to relieve the pain.

Examination with a speculum will show the characteristic ulcer. Complete examination must not be neglected in order to rule out the many possible causes and complications. Allergy study is always indicated.

## 434 Different Parts of the Gastrointestinal Tract

Cancer can usually be ruled out if one is sure that a good specimen was obtained. Serological tests will rule out syphilis. A positive Frei intradermal test is usually considered a reliable indication of the disease but it remains positive for many years and may lead to an erroneous diagnosis in a patient who has later suffered cancer. However, a Frei test is an excellent routine test to perform in any ulcerative rectal condition.

### Treatment

Various antibiotics and sulfonamides often used in combination have been found effective in slowly eliminating the infection and clearing up many of the lesions. However, cases which have been neglected until deformities, fistulas and strictures have developed may require subsequent surgery.

### MYCOTIC INFECTIONS

*Moniliasis* infection with the yeast fungus *Candida albicans* has been considered one of the causes of persistent diarrheas following antibiotic medication. It may produce lesions resembling those of ulcerative colitis. The best treatment consists in giving Mycostatin in tablets of 500,000 units three times daily for four or five days. The same drug is used in combination with broad spectrum antibiotics when these are indicated in an effort to prevent moniliasis.

*Actinomyces*, *histoplasmosis* and other mycotic diseases may affect the rectum directly or may produce lesions caused by sensitivity to the fungus. The whole subject is discussed in the chapter on Mycotic Diseases (pp 623-618).

### PARASITIC DISEASES

Parasitic diseases usually affect the rectum in varying degrees. Amebic ulcers are frequently found in the rectum and sigmoid. Pinworms *Ascaris lumbricoides* are often found only in the rectum causing intense itching inside and outside. *Schistosomiasis* is best diagnosed by rectal biopsy. Other parasites are found in the rectum but do not affect it specifically. The whole subject of parasites is discussed in the chapter on Intestinal Parasites page 134.

### Fissure in Ano

This is a small ulcer at or below the anorectal line at any point of the circumference of the anus but most frequently in the midline posteriorly. It is usually round or slightly irregular but appearing oval or slitlike because of the compression of the sphincter. At its lower end there is often a small protrusion due to edema often erroneously called the "sentinel pile." A new fissure is usually superficial and sharply

defined with an elastic smooth base. As it becomes more chronic it may extend into the subcutaneous tissue even into the muscle becoming indurated and occasionally undermined as a result of infection. It is often accompanied by pruritus ani. The associated severe sphincter spasm causes it to be extremely painful. The pain at first occurs mostly during defecation later becoming constant and extremely debilitating. The pain may radiate into the bladder and genitals upward into the rectum and back and downward into the legs. Retrostaltic symptoms are at times so pronounced that an upper gastrointestinal lesion may be suspected. Women who are too bashful to mention anal symptoms for fear of local examination have at times been subjected to exploratory laparotomy. Insomnia due to pain general irritability and neurosis are common accompaniments.

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### Diagnosis

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Examination with a speculum will show the characteristic ulcer. Complete examination must not be neglected in order to rule out the many possible causes and complications. Allergy study is always indicated.

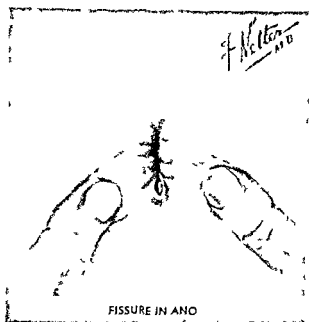


Figure 83 Oval ulcer at anal orifice with sentinel pile at lower end (From the Ciba Collection of Medical Illustrations by Frank H Netter M.D. Copyright Ciba Pharmaceutical Products Inc.)

### Treatment

Surgical treatment except in severe complicated cases can usually be avoided. Although incision or excision may cure the ulcer for the time being recurrences are frequent especially in the allergic cases. Also injuries at operation may result in deformities and strictures.

*General Treatment* This should consist in attention to general hygiene treatment of any general or specific diseases elimination of food allergens from the diet and avoidance of or desensitization to other allergens such as inhalants chemicals or drugs.

The diet should not only be well balanced and nutritious with supplemental mixed vitamins and minerals but also should contain sufficient bulk to produce well formed stools which will stretch the sphincter adequately. The anticonstipation diet described on page 323 is suitable. For a time a small dose of mineral oil  $\frac{1}{2}$  ounce at bedtime and retention enemas of 4 ounces of warm mineral oil at bedtime may be of value. Hemorrhoidal suppositories may be used in place of the oil enemas at bedtime to soothe and lubricate the rectum and anus as the stool passes.

*Local Treatment* Hot sitz baths may be useful in softening the chronic indurated ulcers. Local applications may promote healing. A simple technique should be used as follows:

The speculum lubricated with an analgesic cream is gently inserted

opened and held open so as to expose the fissure. A hemorrhoidal suppository is inserted higher up. The fissure is then dried with a cotton swab. Batyn sulfate solution 2 per cent is applied to the fissure and the surrounding mucosa and allowed to remain for several minutes until anesthesia has been produced. It is then dried off and a touch of silver nitrate solution 10 per cent is applied with a swab to the base of the ulcer only and allowed to dry. Some inert powder is then applied. Two or three such applications at intervals of five to seven days will often produce healing in simple fissures.

Relief of sphincter spasm may be accomplished by the treatment outlined above, the speculum acting to relieve the spasm. At times forcible but gentle stretching of the sphincter with the speculum may help. Sometimes merely insertion of the lubricated finger and holding it in for a while will be sufficient. Leaving in a rectal tube or even a catheter for an hour or more may tire out a spasm. In severe spasm it is occasionally necessary to do an actual division of the sphincter as shown and described in the illustration (Fig. 84). One such division together with local treatment will often promote rapid healing.

### Prognosis

Though local and general treatment will often produce immediate cure, the tendency to recurrence is a disturbing factor. When allergy can be proved to be the cause, treatment for this will prevent recurrence.

## Rectal Fistulas

### ANORECTAL FISTULAS

Fistulas in ano are abnormal tracts leading from the rectum to the skin or to neighboring organs or tissues. They occur frequently in tuber-

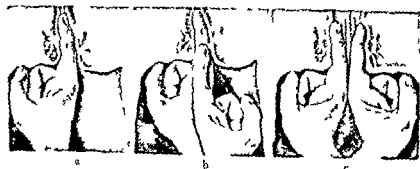


Figure 84 a First step of division; index finger has been introduced into the lower rectum. b Second step; Index finger of the opposite hand is now introduced using the other as a guide. c Third step; Both index fingers assume a similar position in the anal canal. With the wrists crossed the sphincter muscles are stretched by circular movements of the fingers. (Bacon: Anus, Rectum, Sigmoid Colon, 3rd ed., J. B. Lippincott Company.)



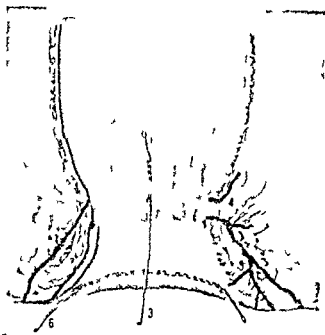


Figure 85 1 and 2 External fistulas 3 complete internal fistula 4 blind internal fistula 5 branching type of fistula 6 horseshoe fistula (From the Ciba Collection of Medical Illustrations by Frank H. Netter M.D. Copyright Ciba Pharmaceutical Products Inc.)

culous patients and may be caused by the *Mycobacterium tuberculosis*. Those opening through the skin are the result of neglected abscesses or of infected operative wounds. The burrowing of an abscess through the loose tissues around the rectum may produce only one opening where it burst through the skin or was incised or there may be networks of fistulous tracts leading through several widely separated external openings. These openings may occur for some distance around the anus even as far as the sacral region, the gluteal region and the scrotum. There is usually only one internal rectal opening. Rarely there may be no external opening with only one blind fistula opening into the rectum or a fistula with two openings into the rectum. There is usually induration in the region of the fistula and more or less deformity caused by the openings. The area around them is often red, macerated and bleeding and there exudes from the openings an irritating, bad smelling, sticky discharge.

### Symptoms

The symptoms are those of the preceding abscess with relief after it opened followed by continuous or intermittent perianal pain and discharge from the opening. When intermittent pain usually follows blocking of the exit causing recurrent abscess formation followed by discharge.

when it opens. Further spread with the formation of multiple openings causes more or less discharge and pain in each. Pruritus is usually complained of and there may be pain on defecation. The chief complaint of the patient is usually about the discharge which soils the clothing causing bad odor.

### Diagnosis

The history of abscess followed by discharge is typical. Careful inspection is often necessary to find the external opening which may appear only as a dimple from which a discharge may be pressed out by massage. At times there may be a fine pink membrane covering the actual opening which may have to be scraped off to induce the discharge. When one opening is found others must be looked for. The internal opening is usually difficult to locate. Stretching the anus and rectum with a speculum may show the small opening. A probe gently inserted through the external opening may be seen or felt inside the rectum and the internal opening thus may be disclosed. Multiple tortuous fistulas are difficult to delineate but must be suspected in any long standing case. Dye injections and x ray studies after injection of opaque media are not of much value. A fistulous opening in the coccygeal region may be mistaken for a pilonidal sinus.

### Treatment

There is only one treatment and that is surgical. The entire tract must be laid open, curetted or excised and packed repeatedly to permit it to heal from the bottom. Various operations have been devised to accomplish this. Under local anesthesia weekly incision and scrapings of  $\frac{3}{4}$  inch to 1 inch of the tract at a time using a grooved director as a guide with packing in the intervals is slow but effective as the various ramifications of the tract are easily followed. At a single radical operation even by the most competent surgeon parts of the tract may be overlooked resulting in recurrences. Operations may be complicated by hemorrhage, infection, sphincter spasm or loss of control, prolapse and chronic anal discomfort. Tuberculous fistulas usually large and sluggish and occurring in complicated cases have a less favorable prognosis than the ordinary cases.

### OTHER FISTULAS

Fistulas between the rectum and bladder, urethra, vagina or uterus are serious complications of operations and of infections and neoplasms of the pelvis. They may follow x ray or radium treatment causing burns and sloughs. They are recognized by the fact that feces may be present in the urine and in a vaginal discharge or urine or blood may be found in rectal discharges. Special examinations by endoscopy and x ray may

clear up the diagnosis. Treatment may require extensive surgical procedures although at times such fistulas have been known to heal spontaneously.

### Rectal Stricture

A rectal stricture or stenosis is a narrowing of the lumen of the rectum. Strictly speaking it refers to a narrowing caused by disease originating in the rectal wall although the term is also used for narrowing from external pressure. Intrinsic strictures presenting a ringlike constriction of the whole circumference less than 1 inch in length are called annular; those involving more than an inch in a tubelike manner are called tubular. Congenital strictures or atresias have been discussed under Anomalies of the Rectum (p. 426). The two most frequent causes of acquired stricture are cancer and venereal lymphogranuloma. Both diseases are discussed elsewhere (pp. 447-433). Strictures may also occur as a result of cicatricial contraction due to infection and ulceration of any kind from trauma and from burns as a result of excessively hot rectal injections or of excessive radiotherapy of the rectum or neighboring pelvic organs. All these causes are discussed elsewhere.

### Symptoms

Strictures are always preceded by symptoms of the causative factor. When the narrowing begins it may cause sudden impaction of feces with symptoms of intestinal obstruction or may at first cause frequent desire to defecate and the passage of more or less diarrheal stools or alternating constipation and diarrhea. With ulceration there is bleeding. Usually bright red or recently clotted blood will be mixed with or smeared on the feces or may be seen only on the toilet paper. Bloody mucus may be passed between defecations with or without a little fecal admixture. General physical examination should always be done to discover any possible contributing factor.

### Diagnosis

Abdominal examination may be negative except for distention in tight strictures.

**Rectal Examination.** The finger may detect anal or lower rectal strictures and blood on the finger will suggest malignancy. Proctoscopy will definitely locate the point of narrowing and disclose the nature of the lesion. Biopsy through the proctoscope is important. Cytologic study of scrapings is not uniformly helpful.

**Barium enema x ray.** If the stricture is complete will show only the location of the lower end of the stricture. If not complete the barium mixture may pass through a stricture by carefully observing the caliber of the rectum the length and degree of the narrowed area can be meas-

ured and the appearance of the inner surface observed (See Figure 78 b page 423 ) A smooth indented appearance suggests external pressure as from an enlarged uterus or prostate or a bladder tumor or a pelvic neoplasm A complete barium enema study valuable as it is should not be performed if the narrowing is too small to permit easy flow of the barium since impaction afterward may cause complete obstruction

### Treatment

When the cause of the stenosis has been determined the indications for treatment are clear The treatment whether progressive mechanical dilatation radiotherapy or operation is discussed in the chapter on each of the diseases which cause strictures In general surgical operations are best performed with electrocoagulation (the diathermy knife") and any tissues removed should be carefully examined for possible malignant changes

### Rectal Impactions

In the absence of actual obstruction or stricture the rectum may be obstructed by impaction of feces of barium from an x ray study or from various foreign bodies either ingested as in the case of bran or seeds or inserted from below or by a combination of these factors In primary megacolon fecal impactions in the rectum and sigmoid are commonly found owing to inability to expel feces as a result of deficient innervation

Fecal impaction may occur merely as a result of atony in an old or debilitated patient especially as a result of a nonresidue diet insufficient water intake and insufficient abdominal exercise It may also result from sphincter spasm due to anal irritation from any disease of the anal region

*Barium impaction* occurs frequently in patients who neglect the care of the diet during x ray study permitting excessive dehydration of the bowel content The barium causes the stool to be white or light yellow when mixed with feces

*Impaction from concentrated bulk* added to feedings such as psyllium seeds bran and commercial preparations containing them occurs when such preparations have been taken alone or added in excess to an otherwise nonresidue diet Contributing factors are neglect of fluid intake and other measures to insure adequate defecation

*Foreign bodies* are discussed elsewhere (p 454)

### Symptoms

The patient may not be aware of an impaction until it has existed for some time Feces above an impaction may become liquefied and pass around an impacted mass until by accretion it causes complete ob

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is dangerous because it may spread infection or cause a perforation. Repeated attempts are often necessary to dislodge a large impaction. At times in stubborn cases the mass must be broken up with a curet or spoon through a speculum or proctoscope and the pieces hauled out.

After removal the rectal injuries and congestion caused by the impaction and efforts at removal usually clear up rapidly without further treatment. Oil enemas or soothing rectal suppositories will help in hastening the healing.

**WARNING** No case of impaction should be dismissed upon removal of the mass. The cause must be determined. Many cases of cancer are overlooked when a complete study is not immediately instituted.

### Allergic Manifestations in the Rectum

Allergic manifestations in the anorectal region are not uncommon. Most cases of *proctitis cryptitis* and *papillitis* are due to allergy. *Fissure in ano* (p. 434) is usually an allergic phenomenon although detection of its cause is often difficult. *Pruritus ani* is formidable though it often appears clear up with remarkable rapidity when an allergen causing it is discovered (Fig. 86). *Purpura* with hemorrhages is usually due to food allergy.

#### Symptoms

All these conditions varying in appearance from that of the skin in simple urticaria to herpes simplex, eczema or ulceration are extremely distressing and rapidly produce a severe neurosis or even a psychoneurosis. The pain and the anal irritation and itching become almost unbearable. The allergic reaction aggravates any other existing condition. Hemorrhoids become congested, prolapse may be produced and



Figure 86. Pruritic skin showing a ventuation of the radiating folds, prominence of the sulci and pallor of the integument. (Bacon: *Anus Rectum Sigmoid Colon*, 3rd ed. J. B. Lippincott Company.)

struction Usually however the patient is worried about the lack of adequate bowel movements feels something in the rectum calling for expulsion and *tries repeatedly to expel it* The efforts may cause sufficient congestion to result in bleeding leading to a suspicion of cancer At times with the assistance of suppositories enemas and his finger he may be able to push out a hard mass which may injure the anal orifice or increase congestion in pre existing hemorrhoids causing more or less profuse bleeding By the time the doctor is called the patient is usually exhausted may be having considerable abdominal distention and tenesmus and will show a congested red bleeding anal orifice

### Examination

Rectal digital examination must be made using a local anesthetic ointment for lubrication in order somewhat to control the pain The impacted mass can then be palpated It is usually smooth hard and difficult to indent The finger can be passed all around it Pieces broken off with the finger will identify its constituents A ray examination can be reserved until the impaction is cleared up when a complete study is indicated At times a fecal impaction is the first symptom of cancer of the rectum or sigmoid and may be seen at first only on a ray examination

### Treatment

Small impactions can usually be induced to pass by means of a cleansing enema Some can be expelled after the patient has injected 4 to 6 ounces of water into the rectum by means of a baby syringe Larger impactions sometimes called fecalomas may be softened by warm retention enemas of dioctyl sodium sulfosuccinate using  $\frac{1}{2}$  to 1 ounce of the 1 per cent solution in 4 to 6 ounces of water retained for four to six hours or longer Capsules of the same salt given by mouth in doses of 50 or 60 mg four or five times daily may aid in the process although several days may be required to get results An impaction may at times be delivered like a baby by stretching the rectum and sphincter below it gradually with the aid of a lubricant and trying to hook the finger over the top Sometimes it can be broken up by pushing the mass backward against the sacrum and delivering the pieces When these procedures are too difficult or painful or the mass too large softening the mass by instillation of 4 to 6 ounces of warm oil with the patient on the left side and allowing it to remain at least six to eight hours to work its way above the mass may facilitate delivery The oil can be a vegetable or mineral oil or half castor oil and half other oil Large doses of mineral oil by mouth may also be tried 1 ounce two or three times a day may be given for a couple of days Hydrogen peroxide by instillation has been recommended but

## The Rectum and Anus

### Neoplasms of the Rectum

445

#### BENIGN NEOPLASMS

Benign tumors of the rectum are of the following types epithelial tumors including papillomas and adenomas together called polyps vascular tumors such as hemangioma and lymphangioma and connective tissue tumors including fibroma myoma and lipoma

#### Pathology

Externally verrucous warts although not strictly tumors are usually classified with them Hypertrophied papillae are also spoken of as tumors Rectal polyps may be single or multiple and vary in size and shape They may be pedunculated or sessile and usually grow slowly They may occur at any age even in children They have been found in as high as 20 per cent of autopsies and 15 per cent of proctoscopies They have a tendency to become malignant and may show no evidence of malignant changes except on microscopic study The other types of tumors mentioned are rare in the rectum and are usually mistaken for polyps Gliomas teratomas and dermoid cysts are also rarely encountered Paraffinomas caused by the injection of paraffin for prolapse and hemorrhoids are the only tumors which may get large enough to cause obstruction Barium granulomas caused by barium used for x ray studies and entering deeper layers of the mucosa through injury or ulceration may also at times be confusing Oleomas usually small and multiple are caused by injection of hemorrhoids with oil sclerosing drugs Endometriosis which may involve the rectum is described under Colonic Neoplasms (p 395) Carcinoids which resemble carcinoma and which are now considered invariable precursors of cancer are rarely found in the rectum and are usually mistaken for cancer They are identified from microscopic sections Leukoplakia of the anal canal occurs in aged persons It appears as pearly white elevated or regular plaques which may become eroded

#### Symptoms

Benign rectal tumors may cause no symptoms being often discovered only on careful examination Most of them subjected as they are to constant trauma congestion and surface erosion have a tendency to bleed Pressure from them may cause interference with venous return and frequently causes hemorrhoids which may also bleed and thus mask the true condition further up The tumors may become large enough to give the sensation of feces in the rectum wanting to be expelled resulting in frequent attempts at defecation with no result or the passage of a little blood or mucus Rarely do benign tumors attain a sufficient size to cause obstruction resulting in peristaltic pains and



## 444 Different Parts of the Gastrointestinal Tract

secondary infection may cause abscesses fistulas, and finally even strictures

### Diagnosis

There is no single specific criterion or test by which allergy can be confirmed as the cause. In all cases such as mentioned above allergy should be suspected. By means of allergy test diets and other methods described in the section on Gastrointestinal Allergy (p. 79) it is possible to discover the agent or agents which are the cause of the condition. The final decision rests on the fact that lesions clear up when the allergen is withdrawn and recur as soon as it is added again.

In rectal allergy the *allergens* may consist of (1) ingestants including foods chemicals or drugs especially broad spectrum antibiotics (2) inhalants such as dust pollens and smoke (3) contact allergens such as clothing materials dyes ingredients of powders ointments suppositories and enemas (4) bacterial parasitic or mycotic allergens originating in infections or infestations in any part of the body (5) parenterally administered drugs hormones vitamins vaccines or blood

### Treatment

It is reprehensible to treat any of these anorectal conditions locally without making a complete study to determine whether allergy is the cause. When a condition which has resisted all kinds of previous treatments clears up promptly upon removing a food such as milk from the diet discontinuing a drug such as phenobarbital or an antibiotic or removing an infected tooth or infected tonsils it is a tremendous satisfaction to the physician and makes the patient forever grateful.

Some of the rectal conditions due to allergy do not clear up completely at once. The proctosigmoiditis resulting from antibiotics may take six to eight weeks or longer before clearing up entirely. Meanwhile local treatments such as instillations ointments or suppositories may aggravate the condition because the patient may be allergic to one of the drugs present in ointments and suppositories usually recommended for such conditions. A persistent fissure may require surgical treatment. Operations have been advised for pruritus. A complete allergy study in addition to helping the rectal condition may demonstrate to a patient that other conditions in various parts of the body are also due to allergy.

### Prognosis

When allergic conditions are neglected and are allowed to continue over long periods complications such as perforations deformities and even strictures may be produced for which surgical treatment may become necessary.

## Neoplasms of the Rectum

## BENIGN NEOPLASMS

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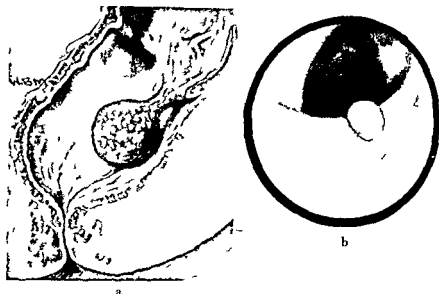


Figure 87 a Polypoid growth of the rectum b Adenoma on the middle valve of Houston Proctoscopic view (Bacon Anus Rectum Sigmoid Colon 3rd ed J B Lippincott Company )

distention Normal stools becoming impacted may become impossible to pass or may be passed in fragments or a diarrhea may be caused by the hyperperistalsis produced by irritation

### Diagnosis

Unless general abdominal findings due to obstruction are present rectal examination is the only method of making the diagnosis External tumors and hypertrophied papillae may be seen by spreading the anal margins or by stretching with a rectal speculum The examining finger may detect tumors which are firm to the touch and may be able to demonstrate their mobility suggesting that they are pedunculated

Proctoscopic observation enables some degree of differentiation (Fig 87 a) The pedunculated tumor of the rectum and sigmoid can be seen Like all other tumors it may be missed on insertion of the instrument but will often pop into view when the instrument is slowly removed Its gross appearance may suggest its nature although this is not always a reliable criterion The sessile tumor must be carefully watched for during the passage and retraction of the proctoscope (Fig 87 b)

Biopsy can be performed on larger or sessile tumors and on leukoplakia or scrapings can be studied for cancer cells Removal of a polyp by careful electrocoagulation of its base is the most certain way of making a diagnosis It must be borne in mind that "cancer in situ" is often present in such tumors even in small ones

X ray studies are of value not only in delineating the size of a large tumor but also in discovering similar tumors in other parts of the colon

### Treatment

The safest procedure is to remove all rectal tumors in order to avoid missing malignant growths or those which might become malignant. Even when a tumor is obviously benign thorough electrocoagulation of its base is necessary to insure that any cancer cells which might be present are destroyed. In the event of the finding of cancer in a removed polyp it requires keen judgment to decide whether radical surgical removal of its site should be undertaken. The fact that cancer is at times cured by electrocoagulation alone must be borne in mind. When for some reason operation is not performed immediately, rectal tumors must be checked at frequent intervals. Endometriosis is best controlled by castration using one of the methods described in the section on Intestinal Endometriosis (p. 395).

### MALIGNANT NEOPLASMS

It is unfortunate that rectal cancer which is comparatively common is frequently overlooked. It usually makes its presence felt early, but the symptoms are too frequently neglected. It acts as a foreign body or a fecal bolus stimulating the desire to defecate and tends to bleed early.

### Pathology

Adenocarcinoma is the usual lesion although scirrhous carcinoma may produce annular constriction and colloid and squamous cell carcinomas may occur. All cancers eventually will ulcerate (Fig. 88). The cancer may involve other pelvic organs. When the whole pelvis is filled with the cancer it is known as a *frozen pelvis*. Metastases to the liver and lymph nodes are found in incurable cancer. It may occur at any age though most often after the age of fifty. Men are more frequently affected. Lymphomas are rare. Carcinoids are discussed elsewhere (p. 378).

### Etiology

Polyps are considered the most frequent *precursors of malignancy* and are not uncommonly found in the rectum. One or more have been found with carcinomatous degeneration at the same time both in the rectum and the colon above. In the anal canal in older patients leukoplakia may be a precancerous lesion. Squamous cell carcinoma can originate in anal skin and in the crypts. Chronic irritation from the laxative habit is a factor. It is also a question whether mineral oil may be carcinogenic.

### Diagnosis

The symptoms are characteristic. Tenesmus increasing in frequency and severity with only expulsion of gas or mucus or nothing at all and

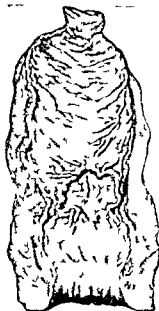


Figure 88 Carcinoma of the rectum showing large crater like ulcer. The rolled edges are well illustrated (Bacon: Anus Rectum Sigmoid Colon 3rd ed J B Lippincott Company)

later blood stained mucus may be accompanied by actual constipation or by diarrhea. The patient often one who has been constipated before feels that the loose stools are a good sign and may not notice blood. Hemorrhoids perhaps present before or due to interference with return circulation by the tumor may actually bleed or may be blamed for the bleeding. Occasionally hemorrhoid operations alone have been done when examination has been inadequate. Attacks of obstipation may be due to impaction of bran or other bulky material taken by the patient for constipation. When such an impaction is relieved in any case a complete examination is called for but I have seen patients get two or three attacks and finally suffer a permanent obstruction before a study was instituted. At times an impaction may be relieved spontaneously by a process of softening and tunneling of its core with resultant diarrheal discharge constituting the familiar alternating constipation and diarrhea often described but really a late symptom. Retrostaltic symptoms anorexia and bleeding gradually produce anemia loss of weight weakness dizziness and finally vomiting. Involvement of other pelvic organs will produce symptoms of female pelvic disease or a urological ailment.

#### Physical Examination

Cancer of the anus can be seen and palpated. Higher in the rectum aside from pallor and evidences of weight loss there may be no defi-

*nite general physical findings* There may be some abdominal distention and borborygmi may be elicited but it is only when obstruction begins that real abdominal distention will occur Because of reflex spasm at Cannon's ring distention may be most noticeable in the right side

*Rectal digital examination* frequently neglected will demonstrate the irregular hard tumor in about half of all cases of rectal carcinoma An anal cancer can be seen on spreading apart the orifice (Fig 89 a) Examination with a speculum or if the tumor is high up through the proctoscope will not only show the characteristic hard bleeding ulcerating nodular or cauliflower like growth but also will permit of biopsy (Fig 89 b) The specimen must be taken from the hard rim of the tumor or ulcer since one from the crater will usually show only necrotic



Figure 89 a Squamous cell carcinoma of the anus Case had been treated for pruritus ani b Proctoscopic view of a carcinoma involving the entire circumference of the rectum Note the cauliflower appearance of the growth (Bacon Anus Rectum Sigmoid Colon 3rd ed J B Lippincott Company )

tissue and will necessitate a second biopsy. Scrapings from the lesion and even rectal washings examined by the Papanicolaou method may disclose cancer cells.

*Vaginal examination* may show spread to pelvic organs.

### X-ray Examination

This is not necessary for making a diagnosis of rectal cancer in fact it is difficult to demonstrate one definitely. However, I have seen cases in which the radiologist made the diagnosis when no rectal examination had been made (Fig 78 a p 423). When obstruction is present only a barium enema is safe and will stop at the obstruction (Fig 79 p 423). If no obstruction is found careful study of the whole colon may disclose other tumors or polyps and a *gastrointestinal series* may show other lesions. Both may also reveal involvement of other areas.

### Differential Diagnosis

In the early stages a diagnosis of ulcerative colitis is frequently made but can be quickly disproved by adequate study. Care must be exercised not to mistake the fecal impaction in cancer for the simple impaction of the aged. Impaction should always be followed by a study to rule out cancer. In the female a pelvic mass felt on vaginal examination must be differentiated from rectal cancer. Bladder, prostatic and ureteral involvement may suggest urological disease until a study reveals the primary cause. Carcinoma of the sigmoid may occasionally cause a prolapse or intussusception and the cancer may then be felt by the examining finger or seen through the proctoscope. The line of cleavage between the sigmoid to which the tumor is attached and the rectal wall will reveal the true situation.

### Treatment

Surgical extirpation should be performed as early as possible. When only the rectum or anus is involved the entire rectum can be removed and a permanent left colostomy established. Attempts at resection and anastomosis to preserve the sphincter and anus are usually unsatisfactory. More extensive involvement may require an operation as devised by Brunschwig consisting in removal of all pelvic organs and implanting the ureters into the descending colon or sigmoid above the permanent colostomy. The discomfort of having urine and feces coming from the same opening usually overrides the advantage of the prolongation of life for a moderate time or the rare cure or failure of recurrence of cancer.

*Palliative surgery* consisting simply in a permanent colostomy may prolong life for six months or more and seems not to inflict too much discomfort if the colostomy is properly cared for.

*Radiotherapy* by x ray radium or radioactive isotopes has been used in frozen pelvis to reduce the size of the tumor so as to permit its removal and postoperatively to prevent spread and destroy any metastases overlooked. As the sole treatment of the cancer it is not justifiable.

*Electrocoagulation* or *desiccation* by means of the so-called diathermy knife may cure small growths it having been contended that the intense heat will destroy cancer cells at some distance from the point of application. I have even seen a couple of patients with extensive involvement practically frozen pelvis cleared up entirely by repeated desiccations with cure for over five years. It is worth trying especially in old debilitated patients who would be poor operative risks.

*Medical Treatment* Preoperatively when the patient is to be operated upon in an emergency for obstruction or perforation the principal preparation would consist in transfusions parenteral feedings of electrolytes amino acids dextrose vitamins and alcohol parenteral injections of antibiotics stimulants as indicated and enemas (except in the case of perforation).

When time permits the patient should be brought up to as good a general condition as possible. This should consist in adequate oral feedings of a balanced highly nutritious diet with added vitamins and minerals adequate fluid to attain a fluid balance and care of any cardiovascular pulmonary or renal complications. Rectal instillations of oil to be retained for softening the stool and later followed by soap suds enemas are worth while. Antibiotics or sulfonamides have been used for prophylaxis given for two or three days before operation but often cause troublesome diarrheas afterward. Transfusions are usually indicated before and during the operation. Excessive bleeding from the rectal lesion can sometimes be reduced by retention enemas of 4 to 6 ounces of 10 per cent gelatin solution given hot at a temperature of 115 to 120° F (never hotter than 120° F).

*Postoperative Care* Early feedings and early ambulation are important (see description under Gastrointestinal Cancer p. 129). Care of a colostomy is discussed in the chapter on Cancer of the Colon (p. 394). When only a palliative colostomy has been done and the rectum with its ulcerating foul-smelling cancer remains oil retention enemas cleansing enemas and the instillation of chlorophyll in solutions may be of much benefit. Increasing doses of sedatives should be used in the later stages. Psychologic care is important (see Cancer of the Colon p. 393).

### Hemorrhoids

Varices of the veins of the superior and inferior hemorrhoidal plexuses are called internal and external hemorrhoids respectively. The name "pile" is applied to both kinds although the term is best applied to the



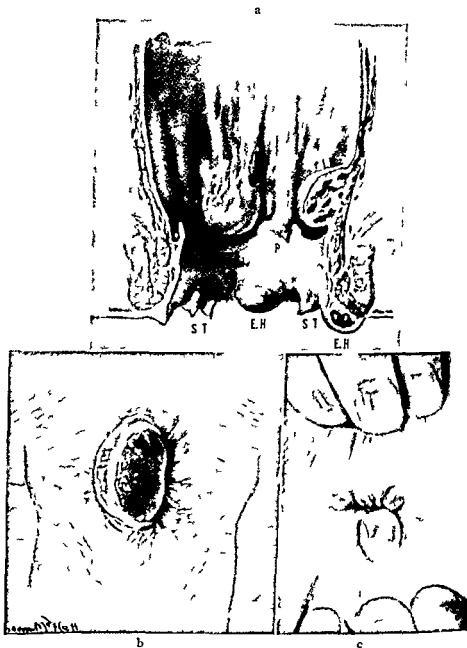


Figure 90 a Hemorrhoids IH Internal hemorrhoids EH external hemorrhoids ST skin tags C cryptitis P papillitis (From the Ciba Collection of Medical Illustrations by Frank H Netter MD Copyright Ciba Pharmaceutical Products Inc ) b Protruding internal hemorrhoid associated with edema of the anal skin c External thrombotic hemorrhoid One of smaller size may be seen above on the opposite side (Bacon Anus Rectum Sigmoid Colon 3rd ed J B Lippincott Company )

smaller external thrombotic hemorrhoids which are covered with skin or lower anal mucosa. Internal hemorrhoids vary in size. They may remain entirely above the external sphincter or may protrude from the anus, often pulling mucosa after them, producing a prolapse (Fig. 90). They may come down only with defecation and recede spontaneously or with the help of the finger, remaining above until the next defecation. Some stay down continuously, coming down as soon as pushed upward. In such cases they enlarge, become congested, may become fibrotic or may develop thromboses or strangulation. Constant slight bleeding may come from surface erosion or sudden more profuse bleeding from rupture of a distended hemorrhoid.

### Etiology

Heredity has been mentioned. The absence of valves naturally interferes with venous return and is aggravated by portal stasis from any cause. Any additional interference will cause hemorrhoids. Conditions causing increased intrapnevic or intra-abdominal pressure, notably that of pregnancy and adiposity, and including tumors of all kinds, are frequent causes. Prolonged standing, horseback riding, sitting especially for too long a time, and straining at stool cause increased congestion. Either constipation or diarrhea may be a cause. Of paramount importance is the fact that cancer of the rectum or sigmoid is a cause of hemorrhoids.

### Symptoms

Itching is a frequent but not specific symptom. A large proportion of internal hemorrhoids cause no symptoms whatever and may be found unexpectedly upon examination. Even a *protrusion* from the anus is frequently overlooked by the patient. *Bleeding*, when present with stools or "on the paper," may be disregarded by the patient as unimportant or its significance may be magnified by the cancer-conscious patient. Discomfort and pain may be caused by persistent protrusion and distention of a large hemorrhoid by thrombosis or by strangulation. *External thrombotic piles* cause a sudden anal pain when a hematoma fills a small vein and produces a tumor of varying size.

### Diagnosis

The symptoms described and the visual demonstration of the nature of the protrusion will usually make the diagnosis. The patient should be examined carefully to determine the causative factor. Cardiac, renal, gastrointestinal, hepatic, urinary, and other possible causes should be ruled out. Prolapse, thrombosis, and strangulation can be seen or felt and can be further observed by means of an anoscope or speculum. Proctoscopy can usually be performed if an anesthetic cream is used for

## 454 Different Parts of the Gastrointestinal Tract

lubrication Inspection is done carefully and gently Complete and repeated proctoscopies and careful x ray study should be done to rule out cancer Too many patients are treated and even operated upon for hemorrhoids without discovery of a cancer which has caused them

### Treatment

Nearly half of the patients have small hemorrhoids which are giving no symptoms and require no specific treatment Many internal and external hemorrhoids will subside and cause no further symptoms if the causative factors are treated or eliminated and a normal diet with sufficient residue and fluids is followed (see Anticonstipation Diet p 323)

*The external thrombotic pile* can at times be disposed of by incision and expression of the clot Excision under local anesthesia is often better especially if much bleeding follows incision Mere incision or even spontaneous remission is usually followed by external tags which usually require no treatment although some patients may ask to have them removed for cleanliness or for fear of cancer

*Internal hemorrhoids* which are large and obviously thrombosed or beginning to be strangulated require some form of operative procedure Small or medium sized bleeding but otherwise uncomplicated hemorrhoids whether prolapsed or not may be treated by injection of sclerosing agents It should not be done in the presence of infection It is not an invariable cure and many patients may require subsequent injections or operation Even operation is not uniformly curative as attested by the many different types of operation recommended

### Foreign Bodies in the Rectum

A great variety of foreign bodies have been found in the rectum Some may have been swallowed although as a rule they are held at one of the narrow parts of the gastrointestinal tract on the way down Some sharp objects however such as nails toothpicks bones and bristles may pass through the intestines and finally reach the rectum at times penetrating its wall Bran has been mentioned as causing impaction Many other swallowed articles have been found

Articles which have penetrated the bowel wall or have been left in at surgical operations may pass down to the rectum These include bullets necrotic bone from the coccyx gallstones surgical instruments and dressings The most frequently encountered foreign bodies found in the rectum are those which have been introduced into it for concealment for punishment or for the purpose of causing erotic sensations I have seen a long packing pencil (Fig 91) hard rubber rectal dilator and a water glass which had been so introduced Instruments used for

examining or treating the rectum such as dilators enema tips even straight proctoscopes may get lost and clinical thermometers are rather frequently introduced too far

A foreign body introduced from below usually proceeds rapidly upward into the sigmoid this upward progress being aided by the efforts at removal either by the fingers or instruments allowing air to rush into the rectum

### Symptoms

The patient may have no symptoms may be unaware that a foreign body is present and may have been unaware of swallowing one Small articles such as seeds may get into the anal crypts causing irritation and infection with the symptoms of cryptitis In the case of larger foreign bodies introduced into the anal orifice and lost the patient is usually terrified and may complain of pains even though no injury has been done With the development of complications such as infection ulceration perforation impaction and later of abscess and fistula the symptoms of these conditions will be complained of further often being a prominent symptom



Figure 91 a Packing pencil in rectosigmoid seen in x ray film as indicated by arrows  
b Photograph of same pencil

## 454 Different Parts of the Gastrointestinal Tract

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fracture of the pelvis occurring principally with vehicular collisions or falls from a height may cause similar injuries

Injuries may be caused by *examination or treatment* Proctoscopic examination frequently causes mild lacerations and contusions Perforation by a proctoscope or by inflation of air during proctoscopy occurs too frequently usually as a result of unskillful use of the instrument although sometimes because of a friable wall as in cancer or ulceration Dilators used by a patient for self treatment or by a doctor may tear the rectal wall even to complete perforation

*Surgical operations* on the rectum or on neighboring organs may cause lacerations contusions and even perforations

*Practical jokes* have been known to shove tools bottles corn cobs and hose nozzles into the rectum even allowing compressed air to rush through a nozzle causing perforations of the bowel

*Radiotherapy* to the rectum or adjacent organs may set up a severe inflammation called "fistular proctitis" which may result in sloughing and perforation of the rectal wall often causing fistulas between the rectum and vagina or bladder

*Lacerations and contusions* due to *internal trauma* from any cause and not resulting in severe hemorrhage necrosis or perforation usually cause little if any discomfort As mentioned under foreign bodies it is remarkable how quickly and completely these injuries will heal without treatment The rectal wall is unusually resistant to infection but occasionally abscesses and their sequelae may result from even mild trauma in a greatly debilitated patient and will require treatment with antibiotics and possibly surgery

### Symptoms

Rectal injuries from *external trauma* may be overlooked when a patient has other more obvious injuries Pain and lower abdominal tenderness may at first be present but may subside until with perforation symptoms of peritonitis supervene Usually blood will be passed X ray films should always be taken to rule out free air in the peritoneal cavity As with internal trauma mild injuries may heal but severe perforation and lacerations will require operation

*Perforation* of the rectal wall results in pain When caused by a proctoscope or a dilator the pain though severe may subside rapidly and spontaneously During proctoscopy in one half of cases perforation can be recognized by seeing and feeling the rent in the wall or by seeing structures recognized as not belonging in the lumen of the rectum or sigmoid such as peritoneal fat and possibly a glistening small intestinal serous coat If not recognized perforation will be followed in six to twelve hours by pain in the pelvis and abdomen by distention rebound tenderness in the lower abdomen and x ray evidence of free air in the

**Examination**

Rectal digital examination may disclose the foreign body if it has not already traveled upward.

Proctoscopy may disclose cryptitis and seeds may be expressed if they are the cause. The foreign body may be seen in the rectum only to disappear promptly as the entering air pushes it into the sigmoid. Upon turning the rectosigmoid angle the lower end of the object may be brought into view.

*X-ray Examination* Scout films will usually disclose opaque objects in the rectum (Fig 91 a). A little barium mixture may show nonopaque objects as areas of decreased density.

**Diagnosis**

In the absence of a history of foreign body ingestion or insertion it is necessary to rule out an acute abdomen, ulcerative colitis, proctitis and possibly cancer.

**Treatment**

Articles felt with the finger may often be delivered through the anus with a little manipulation, the patient assisting by going through the motions of defecation. Objects seen through the proctoscope if small enough may be grasped with long forceps and brought through the lumen. Larger ones must be firmly grasped and pulled down while the proctoscope is withdrawn. Objects thus removed from the rectum if they slip off before being brought through the sphincter usually return promptly to the sigmoid. Sometimes four or five attempts must be made before they can be delivered.

*Operation* If objects are too large or too much impacted to remove from below, surgical removal is indicated.

*Complications* require attention as described in chapters devoted to their treatment.

**Prognosis**

It is remarkable how quickly a rectum which has been badly lacerated by the procedures necessary for removal of the foreign body will heal. Within forty-eight hours such a rectum may show almost no evidence of the injuries sustained.

**Trauma of the Rectum****Causes of Trauma**

As has been mentioned, foreign bodies, even hard stools, may cause direct trauma which may result in varying degrees of injury, infection and even perforation. *External nonpenetrating trauma*, especially with

## SECTION IV

# The Liver and Biliary Tract



peritoneal cavity Under no circumstances should barium be injected into the rectum in such cases When this has been done barium has been found spread all through the pelvis increasing the danger from peritonitis

### Treatment

If *perforation* is recognized immediately and operation performed in the first three to six hours, the mortality rate may be as low as 10 per cent but with delay it mounts rapidly up to 80 or 90 per cent after twenty four hours The rent may not always be found at operation having become walled off with adhesions Breaking them up may result in peritonitis At times it is safer to treat such a patient medically Shock should be combated Bowel distention should be avoided by an indwelling rectal tube by allowing nothing by mouth for forty eight hours and by parenteral feedings of electrolytes blood and antibiotics After forty eight hours rectal instillation of 3 or 4 ounces of warm oil will soften any rectal accumulation and facilitate its expulsion After forty eight or seventy two hours liquid feedings as used in gastric hemorrhage (p 271) can be started and added to gradually so that in ten days the patient is on a full diet indicated for any other lesion During treatment the patient must be watched carefully for evidences of peritonitis development of a mass and any abnormal urethral vaginal or rectal discharges indicating development of abscesses or fistulas Distant embolic phenomena must also be watched for

In the cases described as due to practical jokes the same principles apply as mentioned above The perforation by pneumatic hose is almost invariably fatal

*Strictures resulting from an injury* may require operation or may be treated by gradual dilatation as described on page 440

*Factitial proctitis* has been estimated as occurring in 3 to 5 per cent of cases of uterine or cervical cancer treated with radium or x ray It may be mild and superficial responding in a month or two to treatments recommended for proctitis Deeper burns may cause extensive ulceration and induration with difficulty distinguishable from cancer involving the rectum Biopsy may help in the differentiation There is a tendency to form strictures early so that dilatation with gradually increasing sizes of flexible bougies must be done once or twice a week even though it causes considerable pain and bleeding Failure to dilate may result in complete stricture necessitating colostomy With perforation, due either to the ulceration or to efforts at dilatation treatment as for perforation described above must be carried out

## SECTION IV

# The Liver and Biliary Tract



# The Liver

## General Discussion

### ANATOMY

The liver the largest and most important gland of the body normally weighs about one fortieth of the total weight of the individual. It is a wedge shaped organ with at least five surfaces the shape being due to the fact that the liver is so pliable that it conforms to pressure from the dome of the right diaphragm the rib cage and the surrounding organs. This pliability is also manifest in its various parts causing its lobules and its cells to be polygonal. The liver is covered by a fibrous membrane Glisson's capsule. Its numerous functions make the organ a veritable chemical factory raw material entering it through the portal vein the hepatic artery and the lymphatic ducts. Finished products leave through the hepatic vein to enter the circulatory system for distribution through the blood vessels and lymphatic ducts. Other products travel to neighboring organs some to the thoracic duct. Digestive fluids and excretory materials are excreted through the biliary tract. The liver with its cells arranged in lobules and with its small blood sinusoids ducts and capillaries separated by single layers of connective tissue (which are really extensions of Glisson's capsule) and reticuloendothelial (Kupfer) cells has been likened to a large sponge filled with blood and bile.

### PHYSIOLOGY

The liver is extraordinary in that as much as 75 per cent of it can be destroyed yet the remainder with a little help will maintain life while the regeneration of a fully functioning liver tissue takes place. No specific portion of the liver has been found responsible for a particular function and as yet no definite reason has been found for the fact that death invariably occurs within hours after the entire liver has been removed from an animal or destroyed in man. Much research work continues to be done to find an answer to these questions. Apparently the endocrine glands especially the adrenals play a part in regeneration. New functions are still to be discovered and knowledge of the intricate chemical changes constantly going on in the liver is being augmented constantly. The researches of Mann and Bollman over the past thirty years or more consisting in observations made after destroying or removing the whole or parts of the liver in animals have been of fundamental importance. In the case of partial destruction by toxic agents such as

arsenic phosphorus chloroform or carbon tetrachloride they found that the liver could recover when the poison was withdrawn as long as less than 80 per cent of the functioning liver tissue had been destroyed. Not only are new liver cells reproduced but also the tendency toward intestinal hemorrhage was shown to be overcome by the development of a collateral circulation. An intact portal vein is necessary. Ascites can be controlled by dietary measures especially by the ingestion of a diet rich in carbohydrates and calcium. They have demonstrated repeatedly without having discovered an accurate reason that animal protein especially meat extractives tends to produce early ascites and so cause death in animals that might otherwise survive.

Although it would take a whole book to discuss adequately all the many functions of the liver its principal functions can be grouped conveniently as follows:

### *Excretory Functions*

*Formation and Excretion of Bile* Bile contains not only waste products such as pigments porphyrins cholesterol mucin and inorganic salts but also bile salts essential for fat digestion. The volume of bile estimated as between 500 and 1500 cc in twenty-four hours flows continuously, but its rate and volume of flow and the proportions of its constituents vary at different times of day. They are also influenced by many other factors including the character of a liver disease conditions in the biliary tract the diet the amount of sleep nervous manifestations drugs and by its own or orally administered bile salts which increase volume. The regulation of the flow of bile into the duodenum will be discussed under the functions of the biliary tract.

*Excretion of Alkaline Phosphatase* When excretion is interfered with in hepatic disease there is an accumulation of this enzyme in the blood. The enzyme plays an important part in bone metabolism.

### *Metabolism*

*Carbohydrates* are converted into glycogen and are stored and converted into dextrose as needed to nourish its own cells and maintain blood sugar levels. This function is regulated by insulin and by hormones of the adrenal pituitary and thyroid glands. It may be seriously impaired by liver disease and by disturbances of the endocrine glands. Adequate stores of glycogen appear to discourage fatty infiltration and to fortify the liver against damage from toxic substances. When marked depletion of glycogen is threatened the liver can convert proteins and fats into glycogen. When such depletion is severe a hypoglycemia down to 20 to 40 mg per 100 ml of blood may occur and may result in death.

*Proteins* are stored in the liver and the serum proteins are formed here including serum albumin some serum globulin and all fibrinogen.

and prothrombin. By deamination of amino acids urea and ammonia are formed. With severe hepatocellular damage there is a reduction in serum albumin, a relative increase in serum globulin, a decrease in the blood-coagulating factors, and a decrease in urea and ammonia in the urine and their accumulation in the blood.

**Fats.** At least 60 per cent of fats in the intestine are absorbed by the lymphatic glands, mostly as glycerides, which enter the thoracic duct and are deposited mainly in fat depots throughout the body. The other 40 per cent normally reaches the liver by way of the portal vein, mainly as fatty acids, glycerides of short-chain fatty acids, a little phospholipid, some cholesterol esters, and some reabsorbed cholesterol. Fats are probably not stored normally in the liver cells. Fats and fatty acids are oxidized and ketone bodies are formed, which normally are broken down by the tissues into carbon dioxide and water, thus supplying energy. Other fatty acids may be combined with glycerophosphoric acid and choline to form phospholipids, such as lecithin. Still others are combined with cholesterol to form esters and are absorbed.

Dietary abuses, especially excessive ingestion of fats, carbohydrates, and alcohol, may cause fatty infiltration, as may starvation, diabetes, and other endocrine disturbances, infectious diseases, and poisoning with hepatic poisons such as chloroform, carbon tetrachloride, and phosphorus. Fatty infiltration of the liver is normally prevented, not only by adequate glycogen storage, but also by the presence of certain proteins called lipotropic substances. These include certain members of vitamin B complex, choline,  $B_1$ , and inositol, which apparently are stimulated or formed by the amino acid methionine. In liver disease, excessive amounts of  $B_1$  have been found in the serum and urine.

**Cholesterol.** The metabolism of cholesterol is related closely to that of fats. Although cholesterol is derived partly from food, it is also manufactured by body cells, supposedly the reticuloendothelial cells, some of which are present in the liver. In addition to the liver, the adrenal glands, gonads, and placenta synthesize cholesterol. There is ample proof that it is difficult or impossible to establish a quantitative relationship between the ingestion and excretion of cholesterol. In the blood, cholesterol is found both free and in the form of esters. Whether esterification, the combining of cholesterol with fatty acids, takes place in whole or in part in the liver has not been established. In hepatectomy experiments and in hepatocellular disease, the esters in the blood are markedly decreased and cholesterol tends to accumulate in the liver. Cholesterol in excess antagonizes the lipotropic factors and interferes with phospholipid turnover. In hypercholesteremia, only the complete withdrawal of cholesterol in the diet will reduce the blood level of cholesterol, and such withdrawal will cause symptoms of food deficiency, notably osteoporosis. It is necessary, therefore, that an adequate diet include some

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It is obvious that some knowledge of the many and complex functions of the liver is essential in evaluating symptoms due to its diseases in conducting tests of its functions and in planning treatment

### PATHOGENESIS

The liver can be affected by a large number of agents. These include (1) viruses, toxins, chemicals and poisons of various kinds which will produce a generalized hepatitis, fatty liver or cirrhosis. Hepatic focal necroses may occur in ulcerative colitis. (2) Bacterial infections which may cause liver abscesses and also such specific infections as syphilis, tuberculosis and actinomycosis. (3) parasitic infections of various kinds which invade the liver. *Entamoeba histolytica* may cause abscesses, *echinococcus* may form cysts and various protozoa, nematodes and coccidia will at times cause trouble. (4) Neoplasms, benign and malignant, occurring in the liver, the latter as either primary or secondary lesions. Leukemia also affects the liver. (5) Vascular diseases of both arteries and veins of the liver that can cause serious trouble. (6) metabolic diseases, diabetes, thyroid dyscrasias, amyloid disease and hemochromatosis. Disorders of protein, fat and carbohydrate metabolism may cause liver disturbances or be caused by liver disease. (7) Anomalies and displacements that cause symptoms at times.

### SYMPTOMATOLOGY

There are practically no subjective symptoms which of themselves indicate liver disease; the few symptoms occurring regularly are also present in other diseases. The four most common symptom complexes associated with liver disease are jaundice, ascites, portal hypertension and hepatic coma. Each will be discussed in some detail.

#### Jaundice

Jaundice is due to failure of excretion of bilirubin through the bile into the duodenum and its consequent accumulation in the blood and tissues. According to the mechanism involved in this accumulation, jaundice is usually classified as of three kinds as follows:

*Obstructive jaundice* is due to obstruction in the extrahepatic biliary tract or the small intrahepatic biliary channels. The extrahepatic ducts may be obstructed as a result of calculi, neoplasms, inflammation, parasites or allergic reaction; the intrahepatic through pressure occurring during acute or chronic hepatic disease.

*Hepatocellular jaundice* is due to necrosis or atrophy of liver cells as a result of bacterial or virus infections, organic or inorganic poisons or parasites.

*Hemolytic jaundice* is a rare condition and is caused by excessive destruction of red cells in the body from any cause, by absorption of blood



fat and cholesterol. The conversion of adrenosteroidal compounds to the 17 keto state for excretion in the urine is another function of the liver.

**Vitamins** The production of some and the storage of all vitamins are important functions. The liver manufactures vitamin A from carotene and stores it. It is intimately concerned with vitamins of the B complex and vitamin D. It converts vitamin K into prothrombin.

**Pigments and Other Porphyrins** These are metabolized and excreted by the liver. Hemoglobin has its iron removed and is excreted as *bilirubin* in the bile. It is not normally found in the urine. *Urobilinogen* formed by bacterial action in the intestine from bilirubin is passed in the feces. Some is reabsorbed, enters the portal circulation and is re-excreted by the liver with the bile. A small amount reaching the general circulation is excreted by the kidneys. Normally the urine contains from 0.0 to 3.5 mg of urobilinogen a day. Absence of urobilinogen in the urine means that no bile has reached the intestine. Increase in urobilinogen means that an increased amount of bilirubin is reaching the intestine as occurs in hemolytic jaundice.

*Blood volume is regulated and body heat is produced* by the liver the former by alternate accumulation and expulsion of the water in the liver the latter by the many chemical processes taking place in the liver.

### Detoxification

Detoxification is an important liver function by which the liver protects the body from the effects of many poisons, not only those ingested and reaching it through the portal vein but also those in the circulating blood resulting from inhalation or parenteral administration. Some poisons are chemically changed, oxidized, reduced or acetylated in the liver. Others are excreted in the bile and still others are stored in the liver and fed into the circulation in small amounts. Some are removed from the blood by the action of the reticuloendothelial cells of the liver and the rest of the body. Detoxification is also the mechanism by which the remains of partly used hormones are eliminated. Residual adrenal cortical and pituitary antidiuretic hormones may cause electrolyte imbalance with ascites.

### Formation of Blood

Blood formation is aided by the storage in the liver of supplies of iron, copper and of the antipernicious anemia factors. *Blood coagulation* is regulated by the formation of prothrombin and fibrin to aid coagulation and of heparin to reduce it.

relationships by the scarring and occlusion of blood vessels and lymphatic ducts may result in portal hypertension one of the factors. When disease interferes with the synthesis of blood protein principally albumin resulting in low blood plasma albumin concentration (under 2.5 gm %) osmotic pressure is reduced with resulting edema and ascites. When liver damage interferes with the detoxifying of hormones especially with adrenal cortical hormone and the antidiuretic hormone of the pituitary electrolyte balance is upset sodium is retained and water retention is the result. Ascites is also caused by *exudation* of fluid as a result of increased capillary permeability occurring in peritoneal inflammation and edema of the peritoneum. Chylous ascites results from obstruction to lymph flow as a result of disease involving the mesenteric lymphatics the receptaculum chyli or the thoracic duct or to rupture or perforation of the receptaculum chyli. Rupture of abdominal organs may produce some ascites by *hemorrhage* or the pouring out of cystic fluid.

### Portal Hypertension

Elevation of venous pressure in the portal system is due to interference or blockage of the venous return from the abdominal viscera to the heart. The location of the obstruction may be in the liver above the liver in the hepatic veins or below the liver in the portal system. The back pressure tends to cause congestion in the viscera drained by the portal system causing among other conditions an enlargement of the liver and spleen. The spleen may become damaged hypersplenism may result and this may produce a tendency to hemorrhage in the congested organs. Another effect of back pressure is an effort to relieve congestion by the development of a collateral circulation resulting in varices in the esophagus and hemorrhoids either of which may also bleed.

The *causes* of the three types of blockage mentioned may be summarized as follows:

*Above the liver* the stasis may be due to right sided heart failure to hepatic vein thrombosis pressure from a tumor or cicatrices.

*Within the liver* cirrhosis is the most common cause but any disease causing interference with intrahepatic circulation will produce some portal hypertension. In acute hepatitis it may be transient in abscesses tumors and cysts it will vary according to the size of the lesion.

*Below the liver* the portal vein may be blocked by congenital malformations fibrosis following systemic diseases trauma infection or involvement in neoplasms or adhesions.

**Symptoms and Signs** The symptoms and signs of portal hypertension are those of the primary cause plus those of the complications resulting from the hypertension. Milder degrees of hypertension may produce no symptoms and may be discovered later when severe complications have developed. It is justifiable to consider that any patient with liver disease

from areas of ecchymosis and partially by impaired liver function caused by anoxemia resulting from other causative factors. Examples of this type of disease are spherocytosis, sickle cell anemia and Cooley's anemia.

Other causes of jaundice such as leptospirosis (Weil's disease), yellow fever, parasitic infestation, infectious mononucleosis, hemochromatosis, actinomycosis, leukemia, amyloidosis, tuberculosis, syphilis and allergy must all be considered in making a diagnosis.

**Symptoms** Although varying according to its cause, the symptoms usually include the following:

**Icterus** the yellow staining of skin, mucosa, sclera, urine, sweat and body tissues, which when severe may become dark and greenish.

**Generalized pruritus** which may precede visible staining of the skin. It is not present in hemolytic jaundice. It is thought to be due to bile salts and acids irritating the skin. It may also result from vitamin deficiency.

**Retrostaltic symptoms** may precede or coincide with the onset of jaundice and include anorexia, epigastric "unrest" or distress, nausea, sour eructations or regurgitation and excessive belching. In severe cases, nausea and vomiting may occur and pain may be present in the right upper quadrant of the abdomen.

**Toxic symptoms** include malaise, weakness, mental dullness and headache. Chills and fever may occur even without actual infection. If toxic symptoms are not adequately treated, hepatic coma and death may follow.

**Clay colored stools** caused by absence of bilirubin and excess of fat are an accompaniment of jaundice of the obstructive type, are less noticeable in the hepatocellular type and do not occur in hemolytic jaundice.

**Bleeding** from slight trauma or spontaneously from the mucosa or under the skin will occur when the production of prothrombin, fibrinogen or platelets is interfered with or there is a deficiency of vitamin K.

### Ascites

Ascites is a most important complication of liver disease, particularly of portal cirrhosis. It may be suspected when there is a rapid gain in weight despite evidences of an increasing emaciation of the face, neck and chest. Ankle and leg edema may be the first finding noted by the patient since an enlargement of the abdomen is often considered to be only fat. There may be a considerable amount of excess fluid in the peritoneal cavity before ascites can be determined definitely. The familiar signs of dullness in the flanks, shifting dullness and fluid wave should be searched for carefully.

Ascites, aside from occurring in liver disease, may be a complication of many other diseases. In liver disease, the *transudation* and accumulation of peritoneal fluid have been shown to be due to three main factors: interference with the liver's function of maintaining vascular pressure

known whether all cases are due to the same cause. The findings in blood protein, amino acids, electrolytes and ammonia and in cerebrospinal fluid pressure and bilirubin vary in different cases. High serum bilirubin and low alkaline phosphatase levels are usually but not always seen in fatal cases. Renal insufficiency appears to play a part. Bollman's experiments in animals have shown that coma does not necessarily follow removal or destruction of large parts of the liver.

It is always important to differentiate hepatic coma from other kinds of coma. The coma of Wernicke's syndrome is very similar. It is ascribed to a *hemorrhagic polyencephalitis* due to a nutritional deficiency, an avitaminosis especially of thiamine, niacin and other members of the B complex. It is frequently seen in alcoholic and in old cirrhotic patients. In true hepatic coma, fetor hepaticus, a sweet, musty odor like decayed fruit, is a fairly constant symptom, and electroencephalographic patterns are fairly characteristic. There seems to be no constant finding in patients dying of coma. The only encouraging fact about hepatic coma is that with modern treatment more patients are being saved.

### Other Symptoms

Symptoms other than jaundice, ascites, portal hypertension and hepatic coma are not regularly connected with liver disease. Pain or tenderness in the liver region, most commonly due to sudden distention or actual rupture of Glisson's capsule, may be due to many other causes. Retrostaltic symptoms are often present but are common in most gastrointestinal and many other diseases, as are anorexia, weight loss, constipation, diarrhea, acholic fatty stools and dark urine. Many patients die from acute hemorrhage resulting from mucosal erosions or varicosities. Some have pancreatic necrosis. A history of alcoholism, of exposure to liver poisons, of parasitic infestation, of cancer elsewhere or of recent acute gastrointestinal infection points to the possibility of liver involvement from these sources. In cardiac decompensation a congested liver may produce portal stasis with resultant gastrointestinal symptoms from congestion.

Unfortunately, because of the dearth and inconstancy of symptoms, many cases of liver disease are overlooked until serious complications, even coma, ensue. Study of these conditions reveals the underlying liver disease. It is therefore necessary to be on the lookout for hepatic disease when any of the foregoing symptoms or conditions are found.

### COMPLICATIONS

Disease of the liver may cause complications elsewhere, as a result of impaired function, malnutrition, anemia, portal hypertension, jaundice, pressure from enlargement, metastasis from cancer and sepsis from abscess. These complications are discussed under the various diseases in which they occur.

must have some degree of portal hypertension. When symptoms do occur one of the results of the congestion may be a splenomegaly with its symptoms of hypersplenism including neutropenia, thrombocytopenia, pancytopenia and secondary bone marrow hyperplasia. The congestion of the gastrointestinal tract may cause various gastrointestinal symptoms such as retrostaltic symptoms, bowel disturbances and anorexia with resultant malnutrition. The symptoms of change in collateral circulation are those of esophageal varices and hemorrhoids with hemorrhages, caput medusae and prominence or actual distention of the superficial veins of the abdominal wall. The part played by portal hypertension in producing ascites has already been discussed.

The *diagnosis* of portal hypertension is based on the findings of the conditions just mentioned. Actual measurements of the pressure in the portal vein have been made but are only of academic interest. The blood pressure in the spleen determined by inserting a hollow needle between the lower ribs into the spleen pulp and measuring the pressure of the blood seeping out has been suggested as a valuable method of measuring portal hypertension. Persistently high portal pressure indicates the possibility of hemorrhage from varices and the need for surgery.

### Hepatic Coma

Hepatic coma, sometimes called cholemia, although it may occur without jaundice, is an ominous complication of liver disease and is frequently terminal. It can occur in any form of liver disease but is most common in severe or prolonged cirrhosis and hepatitis. It may occur after operation for obstructive jaundice or after any kind of major surgery in a patient with chronic liver disease or acute hepatitis. Severe bleeding, acute infections of various kinds, acute alcoholism, sudden removal of ascitic fluid by excessive use of mercurials and ammonium chloride or of Diamox or by paracentesis, excessive use of narcotics and sedatives, or shock and anoxemia may precipitate coma. I have seen coma and death follow intravenous injection of fat emulsion in a patient with liver disease.

*Symptoms.* Early symptoms of hepatic coma must be looked for in any patient with hepatic disease. The first symptoms usually consist in drowsiness, mental dullness and carelessness about personal habits followed by incoordination, confusion, restlessness or delirium. Tremors or clonic spasms of the extremities may occur followed by a deep coma resembling quiet sleep. Oliguria or anuria is frequently present. Fotorhecticus can usually be observed. The patient may die in coma in a day or two or may live a month or six weeks before death. Spontaneous remissions may occur and may be transient, much less frequently permanent. The treatment used has been given the credit for those who have recovered.

*Etiology.* The exact cause of hepatic coma is not known nor is it

*Metabolic diseases* causing increase in size include fatty liver cirrhosis hemochromatosis hepatolenticular degeneration lipidosis glycogen amyloid and thyroid disease and diabetes

*Cardiovascular diseases* include passive congestion in cardiac decompensation hepatic artery aneurysm and petechial hemorrhages

*General diseases* associated with hepatomegaly include various infections parasitic and mycotic diseases as well as collagen disease

The size of the liver varies in different diseases and in the same disease at different times With successful treatment of some causes the enlarged liver may decrease to its normal size Rapid shrinkage to below normal size occurring in fulminant acute hepatitis (acute yellow atrophy) and in the late stages of cirrhosis is a bad sign

The size of the liver is not always easy to determine The patient should be examined while he is lying on his back relaxed Percussion is often unreliable In general liver dullness should normally extend upward to the fifth intercostal space in the midclavicular line and downward to the right costal edge or at times a little lower Pulmonary emphysema will mask the upper border and abdominal distention or ascites the lower Palpation of the lower edge of the liver is also difficult Normally the liver edge should not be palpable under the right rib margin even with deep inspiration A large spastic right rectus muscle may be mistaken for the liver An enlarged gallbladder a mass in the region of the pylorus duodenum and biliary tract or even in the colon or an enlarged kidney may be deceptive Ascites will usually prevent palpation until after paracentesis or diuresis has reduced the amount of fluid A narrow thoracic cage or relaxed hepatic supportive ligaments with liver ptosis may be mistaken for enlargement In such cases percussion of the upper border and palpation of the lower border will show that the liver is low on raising the foot of the table or bed the liver may be felt to ride upward A ray examination may be a valuable supplement to palpation and percussion in estimating the size of the liver Its value is discussed later (p 479)

Aside from size palpation of the liver discloses its *contour and texture* An enlarged firm smooth liver is found in fatty liver passive congestion and biliary stasis A hard liver may be due to fibrosis cellular regeneration or cholangiolitis a very hard one to cirrhosis or cancer An irregular or nodular hard surface or liver edge occurs in cancer The nodularity of cirrhosis is rarely discernible Tenderness may occur in almost any type of liver disease It is most frequently noted in cases in which the liver has enlarged rapidly distending its capsule as in infectious hepatitis passive congestion fatty liver cyst abscess or cancer Tenderness is apt to be most severe in rupture of its capsule or of cysts or abscess when it may be interpreted as due to an acute abdomen (p 59)

## PHYSICAL EXAMINATION

A complete physical examination is imperative. Evidence of lesions caused by liver disease or acting as causative factors may be found anywhere. The skin must be observed for mild icterus, increased pigmentation, telangiectasis, spider angioma, xanthoma, varicosities and ecchymoses. Adenopathy should be looked for carefully. The eyes may show an early icterus of the sclera. The mouth, nose and throat may show icterus of the mucosa or evidence of bleeding, or may be the seat of focal infections. Lung cancer and tuberculosis may involve the liver. Cardiac decompensation and coronary occlusion may produce a congested, tender liver. The patient's cardiovascular apparatus requires careful evaluation to determine its ability to withstand operation or long continuing hepatic disease.

*Abdominal examination* may disclose an enlarged liver, the size, contour, texture and tenderness of which it is important to note. The significance of these observations is mentioned later. An enlarged spleen may be felt in portal hypertension. Examination of the remainder of the abdomen for tenderness, rigidity and masses is important. Hernias must be looked for. Rectal digital examination may disclose hemorrhoids which may be secondary to portal stasis or to cancer. Proctoscopy may show varicosities higher up, evidence of recent mucosal bleeding, submucosal ecchymoses or purpuric areas. The male genitals may show scars of chancre, a varicocele or prostatic enlargement. Evidences of infection or cancer of the urinary tract should be looked for. Female pelvic examination may disclose infection, neoplasm or displacement. A Krukenberg tumor due to pelvic implantation of cancer cells from above is of great significance. In ascites, fluid may be felt in the pouch of Douglas. The extremities should be carefully examined, not only for skin conditions, reflexes and tender spots, but also for evidences of circulatory disturbances and varicosities.

## Hepatomegaly

Increase in the size of the liver may be due to many different diseases, both *intrinsic* and *extrinsic*. Following is a list of such conditions.

*Intrinsic*—All diseases and even injuries of the liver are at some time accompanied by an increase in its size. Even in acute yellow atrophy there is occasionally initial enlargement. The intrinsic diseases include inflammatory diseases, neoplasms, abscesses and cysts. Some present smooth, some irregular, nodular enlargement. In some diseases the liver continues to grow in size; in others it may decrease.

*Extrinsic*—*Biliary stasis* from any cause, including biliary tract disease, calculi, neoplasms and enlargement of the head of the pancreas by causing bile to back up into the liver, will cause enlargement.

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*In any disease it is important to evaluate the patient's general condition in addition to the study of a particular system or organ.* Laboratory studies and special examinations to supplement the physical examination of the respiratory, cardiovascular, renal and gastrointestinal systems should be carried out as indicated.

### Liver Function Tests

In suspected liver disease there have been elaborated over the years many tests to evaluate liver functions or liver damage. New ones have been invented, touted for a while, then abandoned because they were not of sufficient value. All require great care and expertness in their performance and experience in their interpretation. When we take into consideration the fact that in any given liver disease only a few of the many liver functions may be interfered with and these not always consistently, the difficulty in arriving at a definite diagnosis by even repeated liver function tests can be understood. A prominent clinician has said that *one good feel of the liver is worth any two liver function tests.* Most clinicians now use groups or "batteries" of tests, each one a test for a different function or group of functions in order to assist in making a diagnosis. They are used in efforts to determine (1) whether hepatic disease is present in nonjaundiced patients, (2) to differentiate between possible causes of jaundice, and (3) to estimate the degree of hepatic disturbance from the time of onset until recovery. I shall group the function tests in the same order as I discussed liver functions. Space will not permit a description of the techniques of the tests which may be obtained from standard textbooks.

**Tests of Excretion** **BILIRUBIN DETERMINATION** When bilirubin is not excreted in the normal way, either as a result of liver damage or biliary tract obstruction, it is retained in the blood serum and some is excreted in the urine. The degree of serum concentration of the bilirubin has long been estimated by means of the icterus index, a fairly simple colorimetric test using a potassium dichromate solution as a standard of yellow color. The index of normal serum is considered as 4 to 6 units; over 8 is abnormal; 15 indicates a latent jaundice. In severe jaundice the index may go to 40 or 50 and higher, even to over 200. Other factors staining the bile yellow may be confusing. These include free hemoglobin in a specimen which has undergone hemolysis, carotinemia, a rare condition due to staining from vegetables, and lipemia, another rare condition.

Somewhat more accurate but also more difficult are the serum bilirubin determinations: (a) the qualitative direct van den Bergh test with positive reactions indicating obstructive jaundice, although at times the test is also positive in severe hepatocellular disease; (b) the quantitative

indirect test which measures the total degree of jaundice but will also include the bilirubin of hemolytic jaundice. Total serum bilirubin varies from 0.1 to 0.6 mg per 100 ml. Above 0.8 mg equal to an icterus index of a little over 6 is evidence of moderate hyperbilirubinemia. At 1.6 mg equal to an index of 16 visible jaundice will soon occur. Many clinicians still depend entirely on the icterus index.

URINE BILIRUBIN determination is of great value. The finding of bilirubin in measureable amounts is always an indication of a damaged liver. It may often be positive before actual jaundice can be detected. It is of value as a screening test for liver disease and as a method of determining the degree of recovery from acute hepatitis. It is absent from the urine in hemolytic jaundice. The old and simple foam test is of little value except in obvious icterus but the newer spot tests are reliable. The Ictotest outfit manufactured by a drug house is accurate and takes but a few minutes. As a valuable screening procedure this test should be a part of a routine urinalysis.

UROBILINOGEN is formed in the intestine from bilirubin in the bile and is excreted in the urine and feces. It will practically always be absent from the urine when there is complete biliary obstruction. When there is only a partial obstruction and a damaged liver which is unable to excrete the urobilinogen which has been absorbed in the intestine and has reached the liver through the blood there may be an excess in the urine and a decreased amount in the feces. A simple quantitative test for urine urobilinogen using a two hour specimen expressed in units is positive at 1 plus or over. The complicated twenty four hour urine test is not much used. Fecal urobilinogen formerly done only on four day collections of feces has been superseded by a simpler one day estimation. Temporary obstruction to bile flow causes changes in its concentration. Less than 0.5 mg a day indicates complete biliary tract obstruction. A simple test on a single specimen showing urobilinogen of 75 to 300 mg to 100 gm of feces would rule out complete obstruction.

*Tests of Metabolism* Many tests of metabolism have been devised and subsequently abandoned. For carbohydrate metabolism the sugar tolerance tests were formerly widely used but now rarely although a hypoglycemia is present in diffuse acute hepatic necrosis. The only test surviving at all is the galactose tolerance test formerly popular but of value only at the onset of acute hepatocellular disease. It is a test of the glycogen mechanism. After an all night fast 40 gm of galactose are given orally in 300 cc of water. Urine is collected hourly for five hours and tested for sugar by the quantitative method. Less than 3 gm obtained in the five hour period is considered within the limits of normal. When 4 or 5 gm or more are excreted it is evidence of impairment of liver function. Intravenous injection of the galactose is considered more sensitive. The test has been largely superseded by other tests.

For *protein metabolism* measurements of *plasma protein* and the relationship of plasma albumin and globulin are undertaken. Since other conditions such as multiple myeloma, some infections and dietary and renal abnormalities may affect these findings, they are of interest only when markedly abnormal. The amount of total protein low in advanced liver disease fluctuates so greatly normally (from 5 to 9 gm per 100 ml) that it is of no real value. The relation of albumin to globulin is normally between 1.5 and 3.0 of albumin to 1.0 of globulin, the former averaging from 3.8 to 6.0 gm, the latter from 1.3 to 3.0 gm per 100 ml. A fall in albumin occurring in advanced portal cirrhosis down to 2.0 gm or less causes a lowering of the albumin globulin ratio. In many cases, especially in ascites or impending ascites, a corresponding increase in globulin to 3.0 gm or higher may produce an inversion of the ratio. In other liver diseases, except in severe hepatitis, there may be little if any change in the albumin globulin ratio.

*Prothrombin concentration* in the blood is important in that a deficiency results in a tendency to hemorrhage. Prothrombin is dependent on two factors: its formation in the liver and its activation by vitamin K. The absorption of this vitamin is dependent on bile salts in the intestine. If prothrombin activity is low, parenteral injection of vitamin K, 2.0 to 5.0 mg, should within twenty-four to forty-eight hours stimulate normal prothrombin activity if the liver is normal. Less than 75 per cent of normal indicates disease. This test is not always available, however, since the prothrombin activity may be normal in the early stages of both obstructive and hepatocellular jaundice. In the later stages it becomes depressed and its measurement is of some value as an index of the severity of hepatic disease and of the danger of hemorrhage. Prothrombin estimations are made by measuring with a stop watch the clotting time of the patient's blood mixed with thromboplastin and comparing it with a control. A clotting time which is five seconds or more longer than that of the normal control is definitely abnormal. A clotting time of longer than twenty seconds is always abnormal. If it takes longer than forty seconds, indicating a plasma concentration of less than 10 per cent of normal, this indicates a tendency to hemorrhage.

*The cephalin cholesterol flocculation test*, comparatively simple to perform, requires fresh reagents to be of definite value. Readings are made in forty-eight hours and are reported as from 0 to 4 plus. The test appears to depend on changes in the albumin globulin and lipoprotein fractions of the plasma proteins and is a sensitive indicator of active hepatic disease. A negative reaction is evidence that there is no active disease; a 1 plus may be of questionable value, but 2 to 4 plus can be considered definite evidence of such disease.

*The thymol turbidity and flocculation tests*, like the cephalin test, are not strictly tests of liver function but are indexes of injury to hepatic

cells resulting in changes in the albumin globulin and lipoprotein fractions of the plasma proteins. The *turbidity test* is rapid the response normally of 0 to 4 units is obtained in one half hour. Three units or more indicate impairment of liver function. It is not a substitute for the cephalin test but is of some advantage in that it is rapid and reaches its peak of abnormality at least a week after all symptoms of acute hepatitis have disappeared and may remain positive for weeks or months afterward indicating residual disease. The *flocculation test* is more delicate in that it is rarely positive in the absence of liver cell damage. Results are reported from 0 to 4 plus and any reading over 2 plus is considered positive.

**TESTS OF LIPID (FAT) METABOLISM** **Blood Lipids** The tests for total lipids in the blood are intricate time consuming and subject to gross error. They are therefore rarely performed. The amount of cholesterol in the blood is a fairly good index of total lipids when hyperlipemia is spoken of it usually refers to hypercholesterolemia. It is important to realize that even in the hands of experts the estimation of blood cholesterol is also subject to error so that only high or low values are of diagnostic significance.

**Blood Cholesterol and Esters** Normal total cholesterol values in the plasma average between 140 and 220 mg per 100 ml esters making up from 60 to 80 per cent of the total. Excessive amounts of total cholesterol 300 mg or more and esters below 60 per cent may be found in obstructive jaundice less frequently in mild hepatitis rarely in early cirrhosis. Since hypercholesterolemia also occurs in diabetes xanthomatosis lipoid nephrosis pregnancy hypothyroidism and other conditions it is not of itself of much value. A diminution in the esters is of much diagnostic importance. In advanced cirrhosis cholesterol especially the ester fraction is low. When the liver is badly damaged as in severe hepatitis from any cause and in yellow fever total cholesterol may go as low as 70 to 100 mg per 100 ml with esters entirely absent or not above 50 per cent of the total. It is also low in acute infectious diseases hyperthyroidism inanition and terminal status.

**Fecal Fat** Fat is found in the feces as neutral fat and free and combined fatty acids. The absorption of fat from the intestine is generally considered to be due to the action of bile in (a) activating pancreatic lipase (b) aiding in the emulsification and digestion of fats and (c) forming soluble compounds from fat and fat soluble substances. When bile fails to reach the intestine in obstructive jaundice therefore fats soaps and fatty acids may be found in excess in the feces. It has been shown that endogenous fat and fatty acids may be excreted into the intestine somewhat confusing the picture. Measurements of total fat content of stools over periods of three days are cumbersome and rarely worth the time they take. Fats tagged with radioisotopes are also used in this connection as are vitamin A tolerance tests. A discussion of steatorrhea will be found on page 531.

*Tests of the Detoxifying Function* The *bromsulphalein test* is the principal means of determining this function. Other dyes excreted by the liver have been tried and found undesirable. It has long been known that the *phthalein* dyes are removed from the blood and rapidly excreted by the liver, probably by means of its reticuloendothelial cells. The *bromsulphalein test* has undergone various changes. Today, after an overnight fasting, 5 mg. per kilogram of the patient's body weight are injected intravenously. Forty-five minutes later 8 or 10 ml. of blood are removed from a vein of the opposite arm. By using the old block calibrator, any retention of the dye after forty-five minutes indicates liver dysfunction. When the newer photoelectric method is used, a reading of more than 4 per cent is abnormal. At sixty minutes no dye should be found. This test cannot be used in the presence of jaundice.

**HIPPURIC ACID SYNTHESIS** A test of the ability of the liver to form hippuric acid is made by the conjugation of glycine and benzoic acid. One hour after a breakfast of toast and coffee the patient empties his bladder and is then given 6 gm. of sodium benzoate in 250 cc. of water. Urine is collected hourly and tested for hippuric acid. Approximately 3 gm. of hippuric acid should be excreted in four hours. Less than this is supposed to indicate liver disease. Low values have also been found in nephritis, which must be ruled out before the test is done. A phenol sulfonphthalein test can be done simultaneously for this purpose. Other conditions, such as chronic passive congestion, anemia and cachexia, will also at times give a positive reaction. While it is a sensitive test if these other causes are ruled out, it is not much used today.

*Tests of Other Functions* *Alkaline phosphatase*, an enzyme normally excreted with the bile, its activity is expressed in Bodansky units, 1.5 to 5.0 units being normal in adults, 5 to 12 units in children. The finding of over 10 units is significant, suggesting obstructive jaundice. Values up to 100 units may occur in obstructive jaundice coincident with high *bilirubin concentration*. In *intrahepatic* and *partial common duct* obstruction in chemical (arsenic) hepatitis and in primary and metastatic cancer up to 60 or 70 units have been observed with normal serum *bilirubin*. The newer King Armstrong units give readings approximately three times those of Bodansky units, their interpretation being the same. Exceptions to the relation between this enzyme and *bilirubin* frequently occur and may cause confusion. The findings are therefore not of much help in differential diagnosis.

*Transaminases* (*glutamic oxaloacetic transaminase* or the more recently discovered *glutamic pyruvic transaminase*) and also enzymes are normally present in human serum with an activity of 5 to 40 units. Determination of transaminases is of value in hepatitis. Although they are increased to 80 to 300 units in infectious mononucleosis, occasionally somewhat higher (to 500 to 700 units) in disease or injury to heart muscle.

and kidneys and trauma to skeletal muscles their values in hepatitis infectious or toxic vary from 500 to 2500 units or more. The units are not increased above 300 in cirrhosis, extrahepatic obstructive jaundice or liver metastases unless hepatitis is also present. Very high readings are almost diagnostic of acute liver damage. The test is complicated and requires rather expensive apparatus.

*Uses of Liver Function Tests* These tests of liver damage and liver function are not all applicable to every type of liver disease. It must be emphasized again that a careful history and complete physical examination are the first and most important steps in diagnosis. *Batteries of tests* useful as an aid to diagnosis are used for different purposes and can be grouped as follows:

1. *For screening blood donors* or other patients in order to rule out liver disease: (a) normal icterus index, (b) the presence of urobilinogen, (c) negative combined thymol tests, (d) normal bromsulphalein test, (e) normal serum albumin values, and (f) absence of bilirubin in the urine indicate that there is no liver disease. These same tests are a valuable aid in determining the progress of a case of hepatic disease and the presence or absence of residual damage.

2. If any of these tests give positive reactions but *no jaundice is present*, additional information may be obtained from the following tests for parenchymal disease: (a) serum bilirubin or icterus index showing preclinical jaundice, (b) cephalin flocculation, (c) protein partition change in albumin globulin ratio, (d) prothrombin time, (e) cholesterol and esters, (f) transaminase determination. Marked increase may occur from one to three weeks before onset of jaundice. In jaundice the bromsulphalein test cannot be used.

3. In a *jaundiced patient* the preceding tests are of value, but certain of them apply more particularly to each of the three types of jaundice as follows:

In *obstructive jaundice* the tests most likely to be affected are the excretion tests, with higher bilirubin concentration or icterus index. Low urobilinogen findings in urine and stool and a high alkaline phosphatase level are helpful.

*Partial obstruction* without visible jaundice is indicated when liver function tests and serum bilirubin are normal but serum alkaline phosphatase is high and there is an excessive retention of bromsulphalein.

In *hepatocellular jaundice* the metabolic tests are liable to show abnormalities. In prolonged obstructive jaundice, however, the resulting liver disease will give positive reactions. The most valuable tests are the cephalin flocculation, combined thymol tests, cholesterol and ester estimation, blood protein partition, and the prothrombin concentration determination. The transaminase test is of great value.

In *uncomplicated hemolytic (constitutional) jaundice* the diagnosis is

*Tests of the Detoxifying Function* The *bromsulphalein test* is the principal means of determining this function. Other dyes excreted by the liver have been tried and found undesirable. It has long been known that the phthalein dyes are removed from the blood and rapidly excreted by the liver probably by means of its reticuloendothelial cells. The bromsulphalein test has undergone various changes. Today, after an overnight fasting 5 mg per kilogram of the patient's body weight are injected intravenously. Forty-five minutes later 8 or 10 ml of blood are removed from a vein of the opposite arm. By using the old block calibrator any retention of the dye after forty-five minutes indicates liver dysfunction. When the newer photoelectric method is used a reading of more than 4 per cent is abnormal. At sixty minutes no dye should be found. This test cannot be used in the presence of jaundice.

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## X RAY EXAMINATION

X ray studies are not of great help in liver disease. The size of the liver cannot be determined accurately from the shadow it casts except in marked hepatomegaly owing to the difference in apparent size when it is posed or rotated. The size of the spleen is determined more easily. The effects of pressure on the stomach and intestines seen in gastro intestinal series and barium enemal studies may show displacement by an enlarged liver (see Fig 92 a). In nonjaundiced patients cholecystography can be used to rule out biliary tract disease. In cholecystectomized patients cholangiography may disclose calculus or other obstruction in the ducts. Chest x rays will show elevation of the right diaphragm due to upward enlargement of the liver from abscess or neoplasm.

The liver may be visualized and delineated by making it opaque by the intravenous injection of Thorotrast but the question arises whether or not it is wise to inject the radioactive element thorium that this contains (see Fig 92 b). Filling of the hepatic vascular bed with an opaque substance by means of injections into the portal vein, aorta or spleen is also of help in outlining tumors or areas of necrosis. It is however a risky procedure. Injection of air into the stomach and colon will outline the liver with air and is a simpler and less hazardous method of estimating the size of the liver than by x ray.



Figure 92 a Hepatomegaly 1 Large liver 2 air in fundus (Magenblase) pushed to left by liver 3 gas in colon pushed down by liver b Thorotrast study Liver and spleen shown filled with radiopaque Thorotrast Liver shadow uneven owing to cancer Gallstones seen through liver shadow Spleen enlargement secondary



usually fairly simple but in sickle cell anemia and in congenital hemolytic jaundice gallstones frequently occur and may cause common duct obstruction which will be confusing. Usually there are essentially normal findings in most liver function tests: absence of bilirubin in the urine and increase in total serum bilirubin with little or no increase in direct acting bilirubin. The findings on physical examination and hematologic studies should confirm the diagnosis.

#### OTHER LABORATORY EXAMINATIONS

##### Blood Tests

In addition to routine complete blood cell counts and chemistry the sedimentation rate, hematocrit study, and the blood examinations involved in the specific hepatic tests, the following may be of considerable value: (1) serologic tests to rule out syphilis as the cause of the liver disease; (2) agglutination tests for specific organisms such as those of the dysentery typhoid group; (3) cultures in suspected bacteremias; (4) a search for malarial parasites.

*Gastric Analysis* Fractional gastric analysis may show normal findings but in some hepatic diseases, notably cirrhosis, subacidity or anacidity may be found. Blood in regurgitated bile stained duodenal content suggests ulceration, the blood coming from a duodenal lesion or the biliary tract.

*Duodenal Contents* Duodenal contents obtained by prolonged aspiration and supplemented by stimulation to the biliary tract, which is emptied by instillation of magnesium sulfate solution or fat, may show reduced amounts of bile in partial biliary tract obstruction, none with complete obstruction. In hemolytic jaundice the bile stain will be normal. In cancer blood may be mixed with bile.

*Stool Examinations* The typical clay or putty colored acholic stools cannot always be assumed to be free of bile pigments, since a small amount of pigment may be masked by the presence of an excess of fats and fatty acids. The pigments consist of bilins or bilinogens obtained from bilirubin, which is never present except at times in severe diarrhea. Absence of urobilinogen occurs in biliary tract obstruction with complete suppression of bile pigment excretion by the liver. In hemolytic jaundice the pigment is normal in amount.

Microscopic examination of the stool is used to look for neutral fats and fatty acids, for undigested meat fibers or starch granules, and for parasites and ova.

Stool cultures to determine whether pathogenic bacteria, viruses, fungi, or amebae are present should also be done.

*Cerebrospinal Fluid Examination* In leptospirosis, particularly bilirubin appears in the cerebrospinal fluid earlier than in viral hepatitis. The fluid can also be examined for evidence of syphilis.

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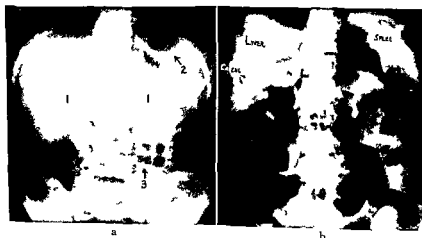


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*Cerebrospinal Fluid Examination* In leptospirosis particularly bilirubin appears in the cerebrospinal fluid earlier than in viral hepatitis. The fluid can also be examined for evidence of syphilis.

2 Conserve liver function by an adequate diet. Such a diet should contain all necessary food elements but should be particularly rich in carbohydrates and proteins. (A sample diet is outlined later.)

3 Prevent hepatic irritation and ascites by avoiding meat and meat extractives. Animal proteins also are undoubtedly implicated in the development of hepatic coma.

4 Protect the liver by giving calcium in some form, not only dietary calcium but also the addition of calcium gluconate.

5 Supply adequate vitamins and minerals, not only in the food but also in the form of multiple vitamin and mineral preparations for oral or parenteral use. Vitamin B complex, the fat-soluble vitamins A and D, and vitamin K should be stressed particularly.

6 Do not eliminate fats from the diet. At least 100 to 125 gm a day should be given, partly in the form of vegetable fats such as oils and margarine. Intravenous injections of fat is dangerous and may cause death in liver disease.

7 There is a question whether or not lipotropic agents should be given in addition to those present in foods. Investigation has shown that with a diet rich in milk, cheese, and vegetables, the lipotropic agents, choline, B<sub>12</sub>, and inositol, members of the B complex group of vitamins, and the amino acid methionine are usually in sufficient amount. In severe hepatic insufficiency, daily doses of choline 2 to 4 gm, inositol 0.5 to 1.0 gm, vitamin B<sub>12</sub> 8 to 10 micrograms, and methionine 1 gm orally or parenterally help to prevent fat deposition in the liver.

8 Maintain biliary tract drainage by frequent feedings and sufficient fat in the diet.

A diet embodying these principles would be as follows:

### Diet in Liver Disease

<i>Breakfast</i>	1 large shredded wheat biscuit (or other cereal) 1 soft egg 1 slice of bread or toast with butter (1 level tablespoonful) Whole milk (1 glass, 8 ounces) Fruit, raw (at end of breakfast)
<i>Mid morning</i>	Gelatin (powdered) 1 heaping teaspoonful mixed with water (1 to 1 glass) followed by Whole milk (1 glass, 8 ounces) or the gelatin may be beaten into the milk
<i>Lunch</i>	Cottage cheese (1 cup) or 1 egg or 1 <sup>1</sup> / <sub>2</sub> package of cream cheese (11 ounces) Vegetables including potato varied from day to day (8 to 8 ounces) Lettuce, large serving with dressing (1 medium with oil-corn, soy bean or peanut) 1 slice bread with butter (1 level tablespoonful) Whole milk (1 glass, 8 ounces) Fruit or gelatin dessert
<i>Mid afternoon</i>	Same as mid morning
<i>Supper</i>	Same as lunch, varying the vegetables
<i>At bedtime</i>	Same as mid morning
<i>Water</i>	At least 6 glasses a day

## BIOPSY

Biopsy can be performed in four ways

1 *An enlarged lymph node* in the groin neck or axilla can be removed and examined for malignant changes or evidences of inflammation

2 *Punch biopsy* or liver aspiration by means of a Silverman needle can be performed. The needle is inserted blindly into the liver and its inner bipronged and grooved needle is pushed ahead liver tissue being trapped in the grooves. Pathologists at first skeptical now feel that they can make a fairly accurate diagnosis from this tissue. In diseases in which parts of the liver are involved as in cancer the needle may not obtain material from the involved portion even on repeated biopsies and a falsely negative report will be made. Although the method is considered safe there is danger of hemorrhage which might require operation to control. In inserting the needle through the thorax there is also danger of spreading infection into the tissues through which it passes. It is necessary to get specific preferably written permission for biopsy.

3 While *peritoneoscopy* is being done specimens can be removed from the liver and spleen or from tumors under visual control and hemorrhage can be seen and stopped. Through the peritoneoscope also a good view of the peritoneal cavity can usually be obtained and an experienced operator can make a diagnosis from observing the gross pathologic condition. Direct cholangiography can also be performed.

4 At the time of operation a wedge biopsy is usually removed but since many lesions are not near the surface a needle biopsy should be performed as soon as the abdomen is opened.

In *summary* it must be realized that a diagnosis of hepatic disease is not easy. At least a combination of detailed history a careful physical examination laboratory tests suitable liver function studies and at times biopsy are necessary to make even a presumptive diagnosis. The complications of the hepatic disease may be so severe and serious as to mask the original condition. It is therefore necessary to be constantly on the alert for possible liver disease.

## TREATMENT

## General Treatment

Consideration of the knowledge of liver functions described heretofore shows that the treatment of a patient with a damaged liver should be essentially physiologic. Such treatment is particularly important in preparation for operation on such a patient. It should embody these principles:

1 Remove if possible any factors which might cause liver damage. This should include poisons toxins alcohol infectious agents focal infections parasites and other diseases which might affect the liver.

by paracentesis must be done with great care. An occasional partial tapping may be helpful. The high protein diet favors absorption of the fluid. Restriction in salt, not a salt free diet, is of help. The use of cation exchange resins may be dangerous.

Operations designed to induce absorption of the ascitic fluid have long been attempted. The shunting operations mentioned for the treatment of portal hypertension will often, by establishing an adequate collateral circulation, alleviate the ascites as well as the esophageal varices. In the past, scarification of the surface of the liver and permitting it to adhere to the peritoneum often accomplished the same effect. An operation to take advantage of the great absorptive power of the small intestinal mucosa has been devised. It has been called ileoentectomy and consists in resecting 15 or 20 cm. of the ileum while leaving the circulation intact and in the peritoneum to increase its vascularity and to encourage collateral circulation. The piece of ileum is then laid open so that the mucosa is exposed in the peritoneal cavity, allowing it to absorb the ascitic fluid. The two cut ends of the ileum are of course joined together. Surgical operations should be performed only if prolonged medical care fails to reduce the ascites.

#### Treatment of Pruritus

The pruritus associated with obstructive jaundice is often difficult to control. All the household remedies have been recommended. Alkaline and starch or oatmeal baths, various antipruritic lotions and ointments, sodium thiosulfate and ergotamine tartrate have all been used with varying success. I have found that the itching is often relieved promptly upon the institution of a diet as outlined, with particular care about the vitamins, calcium and lipotropic drugs. Methyltestosterone, although used successfully in the treatment of pruritus, is dangerous because, in some cases, it may actually increase the jaundice because of its damaging effect on the liver. A new preparation, DHE45 (Sandoz), nontoxic, has been reported as affording great relief.

#### Treatment of Hepatic Coma

Prophylaxis is of most importance and should consist in the avoidance of the precipitating factors (see p. 469). In the early stages, preceding actual coma, an attack may be aborted by a daily diet of about 2000 calories, well balanced except for a slight reduction in protein (75 gm. a day). The addition of large therapeutic doses of all vitamins, especially those of the B complex group, in doses up to 500 mg. of thiamine and 200 mg. of niacin a day is also imperative. Vitamin B<sub>12</sub>, 50 micrograms intravenously, is a valuable supplement. In Wernicke's syndrome, which is due to a vitamin B deficiency and may be confused with hepatic coma, the large doses of vitamins alone will clear up the condition rapidly.

The diet contains approximately 2600 calories with fat 120 gm protein 110 gm and carbohydrate 275 gm. It is good for the average case having sufficient bulk to stimulate bowel function.

The presence of jaundice is not a contraindication to this diet. The caloric value can be raised advantageously by adding extra dextrose as a sweetener or crackers or cake with feedings. For higher protein content an extra egg or extra gelatin may be added. Reduction in these values can be accomplished by removing some of these foods. Alcohol in any form must be forbidden.

#### *Additions to this diet*

**Vitamins and Minerals** One of each of normal vitamin and mineral capsules should be given, larger doses in case of marked deficiency.

**Calcium Gluconate** Doses for calcium gluconate are 30 to 50 grains a day.

**Laxatives** Laxatives should not be required but  $1\frac{1}{2}$  ounce of mineral oil at bedtime may be used temporarily.

**For Complications** If a patient is *unable to swallow* or hold down food from any cause, feedings will need to be given through a duodenal tube or parenterally. Well balanced formulas for such liquid diets are now available in powdered form or in bottles for both kinds of feeding with full instructions for preparing them and even the necessary tubing for their administration. If extra dextrose is required this may be given at the same time. Intravenous injections of fat emulsions must be avoided.

### Treatment of Portal Hypertension

The treatment of portal hypertension is discussed under treatment of esophageal varices in the chapter on diseases of the esophagus (p. 207). The emergency treatment of bleeding varices by the balloon technique does not of course permit of preparation but before one of the shunting operations is undertaken adequate preparation of the patient for operation is imperative. This preoperative preparation should consist of all measures recommended for patients with liver disease (p. 480). In chronic liver disease when esophageal varices have been found even though they have not yet bled, *some form of venous anastomosis such as porto-caval or splenorenal shunt* should be considered seriously as a means of preventing future hemorrhage. The indications for operation and the condition of the patient must be carefully weighed and the patient adequately prepared.

### Treatment of Ascites

If ascites is present the quantity of fluid is restricted and the protein and sugar content is increased. Mercurial diuretics should be used with great caution owing to the danger of inducing coma since they are useful only when combined with the administration of ammonium chloride. For the same reason removal of large amounts of peritoneal fluid

by paracentesis must be done with great care. An occasional partial tapping may be helpful. The high protein diet favors absorption of the fluid. Restriction in salt, not a salt free diet, is of help. The use of cation exchange resins may be dangerous.

Operations designed to induce absorption of the ascitic fluid have long been attempted. The shunting operations mentioned for the treatment of portal hypertension will often by establishing an adequate collateral circulation alleviate the ascites as well as the esophageal varices. In the past scarification of the surface of the liver and permitting it to adhere to the peritoneum often accomplished the same effect. An operation to take advantage of the great absorptive power of the small intestinal mucosa has been devised. It has been called ileoentectomy and consists in resecting 15 or 20 cm of the ileum while leaving the circulation intact and in the peritoneum to increase its vascularity and to encourage collateral circulation. The piece of ileum is then laid open so that the mucosa is exposed in the peritoneal cavity allowing it to absorb the ascitic fluid. The two cut ends of the ileum are of course joined together. Surgical operations should be performed only if prolonged medical care fails to reduce the ascites.

#### Treatment of Pruritus

The pruritus associated with obstructive jaundice is often difficult to control. All the household remedies have been recommended. Alkaline and starch or oatmeal baths, various antipruritic lotions and ointments, sodium thiosulfate and ergotamine tartrate have all been used with varying success. I have found that the itching is often relieved promptly upon the institution of a diet as outlined, with particular care about the vitamins, calcium and lipotropic drugs. Methyltestosterone, although used successfully in the treatment of pruritus, is dangerous because in some cases it may actually increase the jaundice because of its damaging effect on the liver. A new preparation DHE45 (Sandoz) nontoxic has been reported as affording great relief.

#### Treatment of Hepatic Coma

Prophylaxis is of most importance and should consist in the avoidance of the precipitating factors (see p. 468). In the early stages preceding actual coma, an attack may be aborted by a daily diet of about 2000 calories, well balanced except for a slight reduction in protein (75 gm a day). The addition of large therapeutic doses of all vitamins, especially those of the B complex group, in doses up to 500 mg of thiamine and 200 mg of niacin a day is also imperative. Vitamin B<sub>12</sub> 50 micrograms intravenously is a valuable supplement. In Wernicke's syndrome, which is due to a vitamin B deficiency and may be confused with hepatic coma, the large doses of vitamins alone will clear up the condition rapidly.



When coma seems imminent the patient belongs in a hospital. Intravenous injection of 5 to 10 per cent dextrose solution 2000 to 3000 cc per day, with added vitamins may cause a subsidence of the coma if it is not too serious. Electrolyte imbalance frequently a hypokalemia may require up to 80 or 100 mEq of potassium per day intravenously checked by electrocardiographic findings. Infusion of fat emulsions is absolutely contraindicated. Since infection may be a causative factor full parenteral doses of broad spectrum antibiotics are usually given but the diarrhea they may produce is detrimental. To combat the supposed increase in ammonia concentration in the brain monosodium glutamate 20 to 40 gm a day given intravenously has been used with occasional success. Corticosteroids intravenously or intramuscularly are at times lifesaving but must be given with caution and the dosage reduced within a day or two. They have been accused of causing hemorrhage. Recently it has been suggested that the biocatalyst thioctic acid be given intramuscularly in addition to the other agents. As the cause of the coma becomes better understood it is to be hoped that a simpler more specific treatment will be developed.

### Preparation for Operation

Indications for operation will be discussed under each disease. If a patient is to be operated on for a hepatic lesion or when an operation is going to be performed elsewhere on a patient with known or suspected liver disease adequate preparation for operation and care afterwards should include the following:

- 1 Nutrition must be attended to either by the diet outlined previously or if oral feedings are impossible by parenteral feedings. No intravenous injections of fat or oil emulsions should be given.

- 2 Adequate vitamin and mineral supplements as suggested for the treatment of hepatic coma must be provided in order to prevent such a complication.

- 3 Electrolyte imbalance should be corrected or prevented usually by giving calcium routinely and extra potassium as required.

- 4 Lipotropic agents as suggested previously are desirable in order to assure proper metabolism of fats.

- 5 Antibiotics penicillin or broad spectrum antibiotics should be used during the immediate operative period.

- 6 Alcohol should of course be avoided completely.

Treatment of the various specific diseases of the liver will be discussed in the sections devoted to each.

### Anomalies and Displacements of the Liver

The liver does not show as many anomalies as other parts of the gastrointestinal tract and they are of little clinical importance. One

must look out for a left sided liver in *situs inversus*. This is practically always a part of the transposition of the heart, stomach, spleen and other organs and has rarely been found alone. It is of no significance except that if the liver dullness is absent on the right side it may lead to a hasty diagnosis of free air in the peritoneal cavity, a sign of perforation of a hollow viscus. Careful examination will of course obviate making this mistake.

#### MULTIPLE LOBULATION

Multiple lobulation has been described. At best the division of the liver into three lobes is arbitrary. As many as sixteen lobes have been found. The most important extra lobe is Riedel's lobe, usually a projection from the right lobe in the region of the fissure and near the gallbladder. This lobe may be smaller than the gallbladder or much larger and may be mistaken for a palpable or enlarged gallbladder. Depending on whether it is merely an extension of liver tissue from the right lobe or whether it is attached to it by a pedicle it will be more or less movable. It is usually firmer in consistency than the gallbladder. If it is pedunculated a twisting of the pedicle may produce acute symptoms suggesting acute cholecystitis. It is a question whether or not it may cause gallbladder trouble by pressure or traction. This little lobe can undergo any of the changes found in hepatic disease and may be the site of hemorrhages. It is usually only definitely demonstrable at operation or at autopsy.

Differentiation between a Riedel's lobe and the gallbladder and a filled duodenal cap is at times difficult. X-ray scout films may reveal a faint shadow which might be due to either and at times two or three similar shadows may be seen next to each other. If one fills on cholecystography and the one next to it does not fill it may be due to a Riedel's lobe or to a filled duodenal cap. The latter can be differentiated by filling it with ingested barium. When the liver is ptotic the lobe may be low in the abdomen and the palpable mass suggests the right kidney, cecal tumors or even a right adnexal mass. Careful study will differentiate these.

Symptoms are rarely present. Pain, tenderness and fever usually indicate gallbladder disease. A right upper distress has rarely been attributed to the lobe but has usually been associated with hepatoptosis and has been relieved by abdominal support. No other treatment is necessary.

#### HEPATOPTOSIS

Hepatoptosis is usually a part of a general visceroptosis (see p. 106). It is usually found in tall, narrow, asthenic persons, usually female, who have no room for the liver above the costal margin. It is therefore usually a simple manifestation of the asthenic habitus. A wasting disease with

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tively recent years that hepatitis has been shown to be due to a virus infection of the liver parenchyma. Because of its low mortality little autopsy material was obtainable which accounted for the long delay in establishing its pathology. With the large amount of material obtained by biopsy in recent years it has been demonstrated that the lesion consists of a diffuse inflammation starting in the periportal area and involving the cells of the lobules, the reticuloendothelial cells, the hepatic cords and the interlobular spaces resulting in degeneration and necrosis. With jaundice the bile canaliculi become dilated and contain bile thrombi. While all these destructive changes are taking place regeneration of all types of cells is going on.

Recovery of the patient from the disease is dependent not so much on the degree of degeneration as the extent to which regeneration exceeds it. In the fatal cases the infection may be so overwhelming, and liver destruction so rapid that regeneration cannot catch up with it and the patient dies within ten days. Such cases were in the past called acute yellow atrophy, and findings at autopsy have confirmed this opinion. In other cases there may be a contest between degeneration and regeneration for a long time, degeneration finally prevailing and the patient dying in one to three months. In most cases regeneration wins and the patient recovers from the acute stage to remain well or to go on with so-called chronic hepatitis in which regeneration keeps ahead of degeneration but degeneration does not cease. Such cases may develop marked interstitial changes resulting in cirrhosis.

### Etiology

Infectious hepatitis is of two types owing to two distinct filtrable viruses. Man is the only host for these viruses. Virus A (virus IH) is the cause of the one formerly called infectious hepatitis, virus B (virus SH) causes so called homologous serum jaundice or hepatitis. Both viruses have similar effects on the liver although their modes of transmission and their incubation periods differ.

*Virus A* is present in the stool and blood and is usually transmitted through the fecal-oral route although it can also be transmitted by inoculation or ingestion of infected blood of patients who have the disease or have had it recently even in a mild form. Food handlers have often been found to cause epidemics. The incubation period is from two to six weeks. The onset is usually sudden.

*Virus B* not present in the stool is transmitted only by inoculation commonly through parenteral injections of infected blood or blood products or their introduction through infected needles or other instruments. The incubation period is from two to five months, the onset is usually gradual. The extensive spread of this disease during World War II as a result of parenteral injections of immunizing agents, insulin, antibiotics

malnutrition may weaken the ligaments supporting the liver and allow the organ to drop. An abdominal wall that has been weakened by disease by overstretching as in repeated pregnancies or by large tumors may also cause the liver to sag downward. The latter types are seen in later life.

As a rule, no symptoms are attributable to this condition although at times a right upper fullness or dragging feeling which is increased when the patient is in the upright position and is relieved by lying down especially with the hips elevated may be assumed to be caused by it. The symptoms of the generalized visceroptosis especially of nephroptosis may be present. The demonstration of a low lying liver is accomplished by finding the liver edge low down and the upper border low on percussion when the patient is standing and its returning to a higher position when he is lying down. A ray demonstration is of less value since even normally placed livers may appear to be low depending on the position of the x ray tube and the casting of long shadows.

The important point to remember is the fact that a palpable liver edge even much below the costal edge does not necessarily mean that the liver is enlarged. Observation of the body habitus the lowered upper border and evidence of other ptoses should obviate this error. The treatment is that used for visceroptosis in general (p. 106).

#### CORSET LIVER

Corset liver is the name given to a liver ptosis caused by tight lacing which has so narrowed the lower rib cage that there is no room for the liver in its normal position therefore since it is soft and flexible it is pushed downward. Formerly seen mostly in women wearing tight corsets it is now occasionally encountered in men wearing tight belts. Symptoms are absent or are those of ptosis and prophylaxis is obvious.

#### LIVER DISPLACEMENT

Displacements of the liver may be caused by trauma. A large tear in the right leaf of the diaphragm as a result of a crushing injury may permit the liver to be displaced upward. A large congenital defect or hernia may have the same effect. The liver may be pushed downward as a result of effusion or empyema of the right pleura, emphysema or massive pericardial effusion. Subdiaphragmatic abscess may do the same thing. Rarely do any of these displacements cause symptoms referable to the liver.

### Inflammatory Diseases of the Liver

#### ACUTE INFECTIOUS OR VIRAL HEPATITIS

Formerly known as "catarrhal jaundice" and later considered infectious because of its occurrence in epidemic form it is only in compar-

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blood pooled blood plasma and of various other substances is well remembered Allergy skin tests might cause infection The use of parenteral injections by drug addicts and of dirty instruments for tattooing are other methods of transmission Hospital barbers may be implicated This mode of transmission is what makes all blood transfusions and particularly infusions of pooled plasma so dangerous It has been variously estimated that the incidence of hepatitis from blood transfusions is from 1 to 5 per cent from plasma transfusions small pools (ten donors) 1.5 per cent and from large pools (300 donors) 12 per cent The danger of transmission of the disease is present even during the long incubation period and probably for months or years afterwards So far as is known neither gamma globulin prepared by the cold ethanol method nor serum albumin appears to transmit the disease Other fractions notably thrombin have been implicated

### Immunity

An attack of infectious (virus A) hepatitis confers a permanent immunity Since this disease is probably prevalent in childhood and youth often in mild form and so goes unrecognized it is rarely encountered in patients after the age of thirty The length of immunity from serum (virus B) hepatitis is not known A confusing factor is the fact that immunity to either type of virus does not protect from the other When a patient has been infected with one type he may later become infected with the other Cases have been reported in which patients infected with both types at the same time recovered from the early infection of the A type and a few months later had another session after virus B had been incubated

It must be realized that certain occupations are associated with the danger of infection with hepatitis Doctors especially surgeons dentists nurses laboratory personnel especially in blood banks and hospital workers in general are most exposed

### Complications

Complications are common Splenomegaly is a frequent complication showing the changes due to portal hypertension Edema of the wall of the entire intestinal tract is not uncommon occasionally resulting in hemorrhage Phlegmonous inflammation of the ileocecal region occurs in 10 to 15 per cent of severe cases usually associated with ascites and is usually terminal Renal lesions consist of cholemic nephrosis with varying degrees of degeneration Hepatic coma may be fatal At autopsy in such cases even the brain and meninges are found to be inflamed

### Symptomatology

The symptoms of hepatitis vary Although in general the virus A type tends to have a sudden and the virus B type a more insidious onset

there is really little in the way of symptoms to distinguish between the two types. Virus A infection occurs mostly in young persons usually under thirty years of age, is mild in children and is more prevalent in the spring of the year. Virus B infection may occur at any age but is most frequent in the fourth to sixth decades of life.

*Early symptoms* consist in a feeling of malaise, headache, general aches and pains, nausea, vomiting, bad taste, anorexia, dislike of smoking, coated tongue and occasionally some pain in the liver region. There may be signs of an upper respiratory tract irritation and usually fever. This group of symptoms was formerly interpreted as a "cold in the stomach," an acute gastroenteritis resulting from a cold in the head and the icterus noted after several days was believed to come from an inflammation traveling up the common bile duct and causing "catarrhal jaundice." We now recognize all these symptoms as those of the prodromal period of the infection, the preicteric phase of hepatitis. With increased destruction of liver cells and with plugging of the canaliculi, icterus usually develops rapidly; the skin, sclera and urine become bile stained and the stools clay colored. Pruritus is not common. The icterus may clear up rapidly and be hardly noticed or may continue for many weeks or even months. The gastrointestinal symptoms and the fever may subside before or at the onset of jaundice or may continue for weeks. In some cases in which regeneration of the parenchyma is much more rapid than degeneration, no icterus may occur and the true condition may be overlooked unless careful diagnostic study is carried out. In the presence of an epidemic such a study is indicated.

In severe cases complications may occur including gastrointestinal hemorrhages, ascites, acute abdominal pains and evidence of peritonitis, symptoms of renal disease and anemia and hypoglycemia. Occasionally as a result of neglect of rest and diet, overmedication, ill advised surgical operation or serious complications such as those just mentioned, the patient may become drowsy, restless or delirious, have tremors and may pass into a hepatic coma. Without prompt and adequate treatment the patient may die. A considerable number of patients, even though they have recovered from the acute phase of this disease, may continue for years to have some pain in the liver region, some malaise, mild gastrointestinal symptoms and headache. It is difficult in the presence of these subjective symptoms to rule out a chronic hepatitis and these cases are usually classified as such.

### Physical Examination

Physical examination in the prodromal stage may show nothing distinctive although a complete examination should be made to locate any other cause for the indefinite symptoms. There may be an upper respiratory tract infection, a mild fever is common. The first definite finding is



the icterus which may often be seen in the sclera before the skin becomes yellow. A look at the dark urine and light clay colored stools will also be of value. The finding of an enlarged soft cervical lymph node the size of a lima bean low down along the posterior border of the sternocleidomastoid muscle is of some value as a diagnostic aid. On abdominal examination the liver may be just palpable or may extend downward 1 or 2 inches below the costal margin and is usually more or less tender. Rarely an enlarged spleen may be felt. Spider angiomas may be seen on the face and trunk. Hives may occur. Joint pains are occasionally a complaint.

With the development of complications other findings appear. Ascites may come on insidiously so that it should be looked for carefully. Evidences of shock may indicate an acute condition such as phlegmonous inflammation of the ileocecal region or it may precede the passage of blood from a gastrointestinal hemorrhage.

### Laboratory Examinations

Routine examinations include the following

*Blood examinations* are made for the degree of anemia and leukocytosis and for the purpose of ruling out leptospirosis, leishmaniasis, malarial infections, mononucleosis and leukemia. Serologic tests are important. Blood cultures may be required in the presence of prolonged fever. Hypoglycemia may be present in rapid and extensive liver destruction since figures as low as 25 mg per 100 ml have been reported.

*Fractional gastric analysis* does not reveal any constant changes in secretion but the finding of blood in gastric and duodenal contents may be suggestive of one of the complications already mentioned.

*Duodenal contents* which fail to show any bile are significant of either obstruction of the biliary tract or of fairly complete liver destruction.

*Stools* are acholic or show little bile pigment and excessive fat. They should be tested for occult blood and examined carefully and repeatedly for parasites or ova especially in cases in which tropical infestation is suspected. In such cases a complement fixation test for ameba may be indicated. Stool cultures may be of value.

*Urines* should be examined for the degree of concentration which may be masked by a deep bile stain. Bilirubin may be found even before visible jaundice develops. In diabetic patients acetone should be tested for regularly. Porphyrin is found in porphyria (see p. 514).

*Ascitic fluid* should be observed especially for bile stain, for specific gravity and for protein content. A specific gravity of 1.005 to 1.015 and a protein of less than 2.5 gm per 100 ml indicate a transudate. Microscopically there are few cells. With the development of a peritonitis the specific gravity rises to over 1.015, the protein becomes high and cells increase in number. In malignancy involving the peritoneum gross blood

will be seen and malignant cells may show in centrifugal specimens and cell blocks

**Liver Function Tests** Tests of liver function are of considerable value. In the *prodromal preicteric stage* bilirubin may appear in the urine, the icterus index may go up and a positive cephalin flocculation test may be found. Marked increase in transaminase with readings of over 1000 units may occur before these tests become positive. Even the bromsulphalein tests may show retention a little above normal. Bromsulphalein tests should not be done after jaundice has occurred.

During the *active phase* of the disease with jaundice obstructive jaundice may be ruled out by the finding of increased urobilinogen in the urine, a negative direct van den Bergh test and low serum alkaline phosphatase (up to 4 units). Early in hepatitis the alkaline phosphatase may be higher but it subsides rapidly. In severe late hepatitis the van den Bergh test may become positive. The progress of the disease may be evaluated by successive transaminase determinations, icterus index tests, serum and urine bilirubin estimations, cephalin flocculation and combined thymol tests, cholesterol and ester estimations and blood protein and prothrombin tests. If only two or three are to be done, the icterus index, urine bilirubin and cephalin flocculation are the simplest tests and are reliable.

To evaluate the *degree of recovery* from the disease, these tests are of value. It must be remembered that the thymol tests may remain positive long after the disease has apparently cleared up. Others should return to normal before a case can be considered cured. Rapid return to normal of cholesterol and esters is a good sign.

*Failure of complete restoration* to normal of the liver function, called *chronic hepatitis*, can be estimated by the use of function tests. The criteria established by the medical department of the Veterans Administration for a diagnosis of chronic hepatitis are as follows: (1) documented history of acute hepatitis, (2) palpable liver and/or spleen, (3) bilirubinemia—icterus index over 10, (4) thymol turbidity 2 plus or higher, (5) increase in serum globulin (over 3 gm per 100 ml), (6) bromsulphalein retention over 5 per cent in forty five minutes (by photoelectric method), (7) persistent urobilinogenuria or bilirubinuria, (8) liver biopsy by means of the Silverman needle. This examination though risky is of much value in that it makes the pathological diagnosis of hepatitis available. It should however be used with caution, not as a routine procedure. Persistent liver dysfunction is indicated when number (1) and any other four of these findings are positive.

*Peritoneoscopy* is not indicated in an acute condition such as this.

#### X-ray Examination

X-ray studies are not reliable as an indication of the size of the liver.

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diarrhea and fever resembling food poisoning from handling foods and (d) the same precautions as for virus B

2 In *virus B* infection it is important (a) that patients who have ever had jaundice be rejected as blood donors (b) that all hypodermic syringes and other instruments be thoroughly sterilized before and after use even though the patients on whom they have been used give no definite history of jaundice (c) that it be realized that blood transfusions are a potential source of the disease and should be used only when definite indications exist (d) that the use of pooled plasma be avoided

The use of *gamma globulin* as a prophylactic in contacts or as a modifier of the severity of the infection at the onset of the disease is always worthwhile. Little or no protection however is afforded for four to seven days after injection. The single dose required is usually from 0.01 to 0.06 cc per pound of body weight. The protection afforded probably does not last more than a month or two although if the patient has a mild case during that period active immunity will be encouraged.

A vaccine has not yet been developed but with the success attending the use of the Salk vaccine in poliomyelitis it can be expected that one for hepatitis will also be produced.

*Active Treatment* In the *prodromal stage* rest is important. Vomiting can be controlled by autolavage by having the patient drink at one time 16 ounces of warm saline solution (1 rounded teaspoonful of table salt to a pint of water) repeating it if it is vomited and then by giving him a feeding in fifteen minutes. Feedings in accordance with the liver diet (p 481) must be forced to be resumed after each vomiting spell after autolavage. Chlorpromazine and other possibly hepatotoxic antiemetics should be avoided. With the diagnosis suspected this is the time to use *gamma globulin*.

Since no specific therapy is known treatment throughout the disease must be directed to maintaining the nutrition of the patient by forcing the liver diet (see p 481). Parenteral or duodenal feedings may be required as mentioned. Antibiotics and sulfa drugs have not been of any specific value although apparently they do no harm if used for intercurrent infection. Corticosteroid therapy is of no use except in the rare cases with coma. Blood transfusions may be used as indicated especially when shock develops as a result of an acute infectious process or from hemorrhage either of which may be a forerunner of hepatic coma. Lipotropic agents may be helpful in severe cases and can be given in the form of capsules marketed by several drug houses containing the B vitamins choline inositol vitamin B<sub>1</sub> and the amino acid methionine. Alcohol and other hepatic irritants should be absolutely avoided.

Rest has been much stressed as the most important part of the treatment. Prolonged bed rest from thirty to sixty days was enforced in the army during World War II. Careful studies of large groups of soldiers

Although a gastrointestinal series and barium enema study may show pressure from a large liver or spleen they are rarely done in acute disease although they are of value if a cancer is to be ruled out The presence of cancer does not preclude hepatitis Cholecystography is not done in the presence of jaundice

### Diagnosis

The diagnosis of acute viral hepatitis rests on (1) a history of recent (two to six weeks) contact with a case during an epidemic or the history of transfusion or other parenteral injections or instrumentation within a few months before the onset of symptoms (2) the symptoms of acute gastroenteritis occurring at the appropriate interval after such exposure (3) icterus which may however not occur in mild cases (4) acholic clay colored stools (5) a tender and usually more or less enlarged liver (6) positive liver function tests (7) positive findings on biopsy (not usually required)

### Differential Diagnosis

The following conditions must be ruled out in any patient with jaundice

*Toxic or chemical hepatitis* by a history of exposure to poisons or toxins

*Cholangiolitic hepatitis* (discussed in the next chapter p 497)

*Gallstone in the common duct* by (a) history of sudden pain followed by icterus and pruritus (b) absence of urobilinogen in the urine although this may be intermittent (c) positive van den Bergh test (d) liver function tests usually negative except with prolonged obstruction

*Cancer obstructing the common duct* (a) by persistent progressive icterus and pruritus (b) rapid direct van den Bergh reaction (c) rapid downhill course

*Primary cancer of the liver* by a history of preceding portal cirrhosis mild symptoms at first rapid downward course secondarily finding of primary lesion multiple tumors

*Cirrhosis* by the presence of jaundice usually occurring in late stages with ascites In this as well as in cancer biopsy will be of value

Other causes of jaundice must be remembered They are mentioned on page 465

### Treatment

Prophylaxis is most important

1 In *virus A infection* prophylaxis calls for (a) care in the handling and sterilization of feces (b) thorough washing of hands after cleaning the anal region (c) preventing former patients or any patients with

diarrhea and fever resembling food poisoning from handling foods and (d) the same precautions as for virus B

2 In *virus B* infection it is important (a) that patients who have ever had jaundice be rejected as blood donors (b) that all hypodermic syringes and other instruments be thoroughly sterilized before and after use even though the patients on whom they have been used give no definite history of jaundice (c) that it be realized that blood transfusions are a potential source of the disease and should be used only when definite indications exist (d) that the use of pooled plasma be avoided

The use of *gamma globulin* as a prophylactic in contacts or as a modifier of the severity of the infection at the onset of the disease is always worthwhile. Little or no protection however is afforded for four to seven days after injection. The single dose required is usually from 0.01 to 0.06 cc per pound of body weight. The protection afforded probably does not last more than a month or two although if the patient has a mild case during that period active immunity will be encouraged.

A vaccine has not yet been developed but with the success attending the use of the Salk vaccine in poliomyelitis it can be expected that one for hepatitis will also be produced.

*Active Treatment* In the *prodromal stage* rest is important. Vomiting can be controlled by autolavage by having the patient drink at one time 16 ounces of warm saline solution (1 rounded teaspoonful of table salt to a pint of water) repeating it if it is vomited and then by giving him a feeding in fifteen minutes. Feedings in accordance with the liver diet (p 451) must be forced to be resumed after each vomiting spell after autolavage. Chlorpromazine and other possibly hepatotoxic antiemetics should be avoided. With the diagnosis suspected this is the time to use *gamma globulin*.

Since no specific therapy is known treatment throughout the disease must be directed to maintaining the nutrition of the patient by forcing the liver diet (see p 451). Parenteral or duodenal feedings may be required as mentioned. Antibiotics and sulfa drugs have not been of any specific value although apparently they do no harm if used for intercurrent infection. Corticosteroid therapy is of no use except in the rare cases with coma. Blood transfusions may be used as indicated especially when shock develops as a result of an acute infectious process or from hemorrhage either of which may be a forerunner of hepatic coma. Lipotropic agents may be helpful in severe cases and can be given in the form of capsules marketed by several drug houses containing the B vitamins choline inositol vitamin B<sub>1</sub> and the amino acid methionine. Alcohol and other hepatic irritants should be absolutely avoided.

Rest has been much stressed as the most important part of the treatment. Prolonged bed rest, from thirty to sixty days was enforced in the army during World War II. Careful studies of large groups of soldiers

with acute hepatitis in recent years have shown that complete bed rest is not as important as it was thought to be. During the prodromal and early acute stages the patient should have almost complete bed rest and be allowed up only to go to the toilet. After this an hours rest after meals with additional rest if the patient feels sick is usually sufficient. A reduction in transaminase activity is a good index of recovery. Serum bilirubin concentration should be checked at intervals of a week or so until it drops below 15 mg per 100 ml. When after subsidence of jaundice the bromsulphalein test also is 5 per cent or less in forty five minutes and bilirubin has disappeared from the urine the patient may be allowed normal activity but if all four tests show increases in two to four weeks more rest is indicated. Persistently high levels call for individual management. Recurrent abnormalities call for further evaluation perhaps with biopsy. Check ups at six month intervals should be done in order to determine possible cirrhosis.

*Pruritus* is a rare complication and can usually be controlled with diet and vitamins.

*Restlessness* and occasional pain are most safely controlled by aspirin occasionally aided by codeine. Opiates and barbiturates are not well tolerated.

*Ascites* is rare except in severe terminal situations in which paracentesis and the usual measures are of little if any value.

Threatened *hepatic coma* calls for intravenous hypertonic glucose (10 or 15 per cent) solution about 2 liters in the first twenty four hours and one or two units of blood. The use of antibiotics has been recommended on the theory that by sterilizing the gastrointestinal tract they will combat any complicating infections and besides will relieve the liver of the bombardment of endogenous metabolic products. Though only rarely of benefit they will usually do no harm to the liver and may be tried. Monosodium glutamate may be of benefit. The treatment of hepatic coma is discussed in the general section on the liver (p. 483).

When a patient has fully recovered he must be warned against the use of alcohol, the excessive ingestion of meats and exposure to hepatic poisons. A well balanced diet with vitamin supplements will fortify the patient against relapses and will build up resistance to many infectious diseases which might injure the liver (p. 21).

### Prognosis

In most cases of infectious hepatitis whether due to virus type A or B the patient acquires an immunity to the specific virus only. Recurrent attacks are usually due to infection by the other virus and may follow the same course as the original attack. In such cases the possibility of permanent hepatocellular and interstitial changes is enhanced. Some patients may become carriers often after mild even unrecognized attacks.

of hepatitis. The carrier state may last for years making any person who has had hepatitis definitely a dangerous blood donor. Whereas the battery of liver function tests previously mentioned may rule out carriers, a small percentage of donors known to have transmitted hepatitis repeatedly have been found to have normal function tests. It is however wise in evaluating donors for routine transfusion to do at least a few of the function tests to rule out definitely dangerous donors.

### TOXIC HEPATITIS

The term "toxic hepatitis" includes the various forms of liver damage caused by the toxins of acute infectious diseases by drugs and medication by chemicals and by allergic reactions. Some of these causes merge into each other. The liver damage may consist in actual hepatocellular injury with necrosis, may involve only the bile canaliculi, may affect only the interstitial tissues, or may produce a combination of two or all of these forms of injury.

#### Symptoms

The symptoms will depend on the cause of the damage and its extent. Some patients already ill with an infectious disease such as pneumonia, malaria or infectious mononucleosis will have symptoms of liver damage. Some patients who are being treated with a drug which is toxic to the liver or to which the patient is allergic may have insidious symptoms of mild liver disease or may be suddenly precipitated into the symptoms of marked liver necrosis. Others who have previously been well will begin to tire easily, feel weak, have headaches and dizziness, perhaps for months until regeneration of liver tissue does not keep up with degeneration. Then prodromal symptoms of chills and fever, of nausea and vomiting and perhaps diarrhea will occur and be followed after several days by icterus of varying degrees. The subsequent symptoms will often be hardly distinguishable from those of infectious hepatitis or may suggest biliary tract obstruction. In cases in which jaundice does not appear the patient may be treated for neurosis, for gallbladder disease, for irritable colon or for some other chronic ailment for some time before the true situation is suspected.

#### Diagnosis

A careful history will often disclose the cause of the symptoms if liver dysfunction is suspected. Thorough examination and laboratory tests may indicate an acute infectious disease. Careful questioning may elicit the information that a patient has been using drugs or handling or inhaling fumes of chemicals known to be hepatocellular poisons. The following substances are known to cause such damage and I shall indicate how the patient may have come to use them.



1 *Drugs*

*Arsenic* used in some form as a hematinic an amebicide or other parasiticide

*Cinchophen* used for relief of pain in chronic arthritis

*Sulfa drugs* used for infections of various kinds

*Newer drugs* especially chlorpromazine used as an antiemetic and for the relief of pain and anxiety states

*Phenylbutazone*, used for the alleviation of joint and muscle pains

*Para aminosalicylic acid* and *pyrazinamide* used in the treatment of tuberculosis

*Propylthiouracil* and *methimazole* used in the treatment of thyroid dyscrasias

*Methyl testosterone* a valuable adjunct to pituitary gonadal therapy

There is a question whether in the case of each of these drugs *allergy* to the drug may be the cause of the liver damage or whether the action of the drugs itself is the cause. Practically all of them have been used freely on large numbers of patients without doing such harm

2 *Chemicals* There are certain *chemicals* long known to cause actual necrosis of liver cells per se

*Phosphorus* poisoning with which was formerly prevalent among workers in match factories

*Carbon tetrachloride* used for dry cleaning purposes is a solvent for dyes for the coloring of shoes occasionally for black hair dye and as an insecticide. Persons working in cramped poorly ventilated quarters and exposed to this solvent may quickly receive bad liver damage. The same chemical or its close relative *tetrachloroethylene* used to kill intestinal parasites may make trouble for susceptible persons or those having had previous liver damage

*Chloroform* used as an anesthetic or as a solvent and also other halogenated hydrocarbons especially those used as solvents in paints used for spraying

Some *aniline dyes* and food flavors may cause liver injury

3 *Foods* The principal foods causing hepatic damage are certain *mushrooms* and certain *tropical fish* which may cause sudden death from liver necrosis

*Alcohol* must be classed as a liver poison although there is still considerable controversy as to whether alcohol itself does the damage or whether the trouble is caused by the alcohol imbibers taking less food and especially fewer vitamins than required. It is probably true that much damage is done as a result of malnutrition in general and insufficient intake of the B complex vitamin especially choline. It appears that alcohol increases the choline requirement of the body. Mallory demonstrated *cholesterol inclusion cells* small discrete bodies each surrounded by a choline sheath, seen in the liver only in alcoholic patients. In rats definite

hepatotoxic effects have been demonstrated. It has also been proved that alcohol increases the hepatotoxic effects of other liver poisons.

4 *Allergic Reactions* Allergic reactions are discussed under gastrointestinal allergy (p. 79).

### Examinations

*Physical examination* may disclose little or may reveal an enlarged liver and perhaps the spleen also. Laboratory tests will show the findings described under infectious hepatitis (p. 490).

*Punch biopsy* may in some cases show more or less marked hepatocellular damage. In others, as in the case of some of the newer drugs mentioned previously, there will be the changes seen in obstructive jaundice. Regeneration is always going on except in severe fatal cases.

### Survey

A careful and complete survey of the patient must be undertaken in order to determine whether any other damage has been done by the toxic agent. Blood examinations are important to rule out damage to the spleen or bone marrow. Evidences of renal damage should be looked for. In cases in which no history is obtainable the finding of the poison in the blood or excreta would be of paramount value. In rapid and extensive liver destruction hypoglycemia down to 40 mg. or less per 100 ml. may be found.

### Treatment

Elimination of the causative agent is of course the first desideratum. If an antidote is available it should be administered. The liver damage should be treated like damage from any other cause as outlined in the chapter on the treatment of liver disease (p. 450). Unless there has been long neglect of the poisoning resulting in irreparable damage such as cirrhosis, nearly all patients can be expected to recover.

### CHOLANGIOLITIC HEPATITIS

Cholangiolitic hepatitis is occasionally a sequel to infectious or toxic hepatitis although it is rare. It usually occurs a year or more after the acute disease and has been ascribed to obliteration of intrahepatic bile ducts on subsidence of the acute disease. Some cases have been reported in which no history of previous infectious hepatitis or biliary tract disease of any kind could be obtained. In such cases differentiation from infectious hepatitis may be difficult. However, liver function tests are within normal limits except for those showing biliary obstruction (high alkaline phosphatase and total cholesterol reduced or absent urinary urobilinogen). Biopsy shows no hepatocellular damage. The pathologic findings are usually those of a cholangitis with dilatation and neutrophile infiltra-

tion and cholestasis. The resemblance to the changes seen in nephrosis has led to the use of the term "cholangiolosis" for this condition. It is associated with deepening jaundice and the usual symptoms accompanying jaundice. Treatment as recommended for acute infectious hepatitis (p. 492) will often clear up the symptoms within a month or six weeks. In refractory cases corticosteroids have been recommended because of their favorable action in nephrosis. Failure to clear up may result in so-called cholangiolitic or Hanot's cirrhosis, with or without dermal xanthomas.

### LIVER ABSCESS

Because of its many anatomical and physiological factors which tend to combat infection, the liver is much more rarely involved in abdominal infections causing abscess than in abdominal malignancies. Infection or infestation producing abscess may reach the liver through its blood vessels, bile ducts or lymphatics. Infection may result from trauma or from spread by contiguous infections. Abscesses may occasionally accompany systemic mycotic or parasitic infections. In general, most liver abscesses are of two kinds, pyogenic and amebic, and either kind may be solitary or multiple, large or small. In those occurring near the periphery, perihepatitis will occur.

#### Pyogenic Abscesses

These abscesses are caused principally by infection of the portal vein, suppurative *pylephlebitis*, or by infective material carried through an uninfected vein by *septic thrombi*. In both cases the infective material originates in the area drained by the portal vein and its tributaries. Suppurative appendicitis is the most common cause, but other lesions of the gastrointestinal tract, such as dysentery, ulcerative colitis, diverticulitis, peptic ulcer, and abdominal carcinoma or tuberculosis, usually when infected or perforated, may provide a port of entry to the portal system. Suppurative lesions in the biliary tract, infected hepatic cysts or tumors, suppurative pelvic, urinary, splenic or pancreatic disease may also act as foci.

Usually, pyogenic abscesses associated with *pylephlebitis* are multiple, although at the start a single abscess may occur around the infected portal vein, and later abscesses will form along its branches in the liver. The liver enlarges, and with the breaking down of septa between abscesses, larger and larger abscesses are formed until the liver resembles a large sponge which on squeezing (at autopsy) oozes pus all over. Any organism may be the infective agent, although cultures usually show *streptococci* and *colon bacilli*. Final termination is almost invariable.

Infections through the *hepatic artery* are rare and are usually small and multiple, although a few cases of single abscess have been reported.

They are the result of septic emboli carried from infections in any part of the body. In other words they are part of a bacteremia. Retrograde infections through the hepatic vein have been postulated but must be rare.

In abscess *secondary to biliary tract infection* usually suppurative cholangitis the infection spreads along the bile passages into the liver usually producing multiple cholangiolitic abscesses (see p 567).

*Trauma* may produce abscesses in different ways. *External trauma* such as puncture by knives bullets or needles (as in punch biopsy or abdominal paracentesis) may carry infection directly into the liver or the wound may be secondarily infected. *Foreign bodies* such as swallowed needles pins bones glass toothpicks or other articles may puncture the gastrointestinal wall and eventually reach the liver become encysted and subsequently infected. *Distant injuries* causing infected wounds may produce infective foci.

*Contiguous suppuration* in the base of the right lung or pleura (empyema) or below the right diaphragm right perinephric abscess or suppuration secondary to infection or perforation of the biliary tract stomach or duodenum may involve the liver capsule and then the liver itself.

*General infections* such as typhoid fever and influenza have been complicated by liver abscess. Systemic mycoses actinomycosis and histoplasmosis rarely produce abscesses (p 531). Parasites such as flukes and roundworms entering the liver may cause abscess formation. Infected cysts and granulomas are additional causes.

### Amebic Abscess

Amebic abscess usually single and large and occupying the dome of the right lobe is seven times more frequent in men than in women. Frequent in the tropics it is rarely seen in the north. The organisms reach the liver usually through the portal vein more rarely through the lymphatics or the peritoneum (p 139) causing a hepatitis which may go on for a time and subside or may go on to abscess formation. Although it does occur during an acute dysentery in the tropics it is more frequently an aftermath of dysentery in the north. It may occur as late as months or years after an attack of dysentery which is frequently not even remembered by the patient. Abscesses have been found often in ameba carriers who have never experienced an acute attack of dysentery. Observers have reported that in 10 to 50 per cent of cases no amebae have been found in the stools since they may live in the liver for long periods before symptoms develop.

A frequent complication is rupture which may occur upward to perforate the diaphragm into the right lung a bronchus the pleura or the pericardium or may go downward involving peritoneum colon kidney or vena cava. Lung abscess is the most frequent complication. The young

amebic abscess may have a poorly defined abscess wall but older large abscesses may have thick walls. The abscess is filled with a brownish red mush resembling anchovy sauce. The material consists of broken down liver tissue and blood and does not contain pus unless it has been secondarily infected. The trophozoites are usually not found free in the material but can usually be found in scrapings of the lining of the abscess.

### Symptoms

The symptoms of liver abscess of either type are usually those of sepsis with spiking fever, chills and sweats. Pain usually constant and dull may occur early or late, may be over the liver or in the chest, axilla or back. It may be increased by exercise or inspiration is usually not affected by food. Gastrointestinal symptoms are usually minimal unless due to the exciting cause. General weakness, anorexia and weight loss are usually present. With the occurrence of rupture a sudden pain with shock usually occurs followed by chest or abdominal symptoms according to the direction taken by the rupture. In general the patient appears septic and has a sallow skin and a cachectic look. The pulse is irregular and weak.

### Physical Examination

Tenderness and enlargement of the liver are fairly constant, the enlargement being upward or downward. Jaundice is present more in multiple pyelephlebitic abscesses although less than in those due to suppurative cholangitis. Jaundice with a single abscess indicates pressure on the bile ducts. Ascites is an indication of impending dissolution.

### Laboratory Examinations

The findings are those of sepsis. Leukocytosis with marked predominance of neutrophils and a rapid sedimentation rate are to be expected. In long standing cases emaciation and anemia will be present. With development of complications the findings of empyema, lung abscess, pericardial effusion, impaired renal function, hemoperitoneum or peritonitis and colonic findings supervene. Liver function tests would show abnormality only in advanced neglected cases in which the liver parenchyma was badly damaged.

### X-ray Findings

As mentioned before it is difficult to evaluate the size of the liver. Visualizing the liver by means of Thorotrast or by splenoportography will often show up areas of decreased density at the sites of abscesses and may solve the problem of whether or not they are single or multiple. A chest x-ray may show the upward excursion or fixation of the diaphragm (see p. 479). A gastrointestinal and gallbladder study will show pressure from

an enlarged liver and may also indicate a lesion such as was mentioned previously which may have acted as a focus. The chest or abdominal complications following rupture would present definite x-ray findings. In perforation of a hollow viscus free gas or air in the peritoneal cavity would be a diagnostic factor.

### Diagnosis

It is only by careful study and observation that a diagnosis can be established. In the presence of a history of one of the etiological factors mentioned before the development of a septic course with liver tenderness and enlargement should occasion a suspicion of liver abscess. Sudden onset with septic symptoms occurring after suppurative appendicitis or diverticulitis suggests multiple abscesses in association with pyelophlebitis. A more insidious onset with less definite septic symptoms suggests a probable single abscess especially if there is a history of antecedent dysentery. The x-ray is probably the most reliable diagnostic aid but the methods used for visualizing the liver entail some risk in themselves. It is undoubtedly true that the diagnosis is frequently missed. Perinephric abscess, abscess of the biliary tract, perforation of a hollow viscus or a bronchus, typhoid fever, pneumonia, syphilis and cancer have often been diagnosed and often correctly so but with the secondary abscess not suspected. With the development of complications the diagnosis may be even more confusing.

### Treatment

Liver abscesses present a difficult problem. In general the patient should be prepared for operation by a careful nutritious diet such as has been recommended before for a damaged liver (see p. 480). The dehydrated patient should be given ample fluids by mouth supplemented by glucose and saline solution parenterally. Various antibiotics have been used as they have appeared on the market and it has been felt that some of them aborted or at least controlled the extension of the abscesses. They are certainly a help if used before and after operation. In *amebic hepatitis* it has been claimed that formation of abscess has been prevented by the use of emetine hydrochloride 1 grain hypodermatically once a day for not more than ten days. However because of its toxic effect on the heart muscle Aralen and quinacrine hydrochloride are now used. Operation is usually contraindicated. A few aspirations may clear up the condition (p. 189).

*Surgical treatment* includes removal of a part of the liver containing a single abscess or incision and drainage of the single or multiple abscess. Great care must be exercised to avoid spilling infected contents and approach either through the chest or abdomen is chosen with this end in view. The mortality rate is high being highest in pyelophlebitic abscesses.

and lowest in amebic abscess unless the patient is in a very depleted state

### PERIHEPATITIS

Any hepatic disease reaching the surface of the liver may produce an inflammation of Glisson's capsule called perihepatitis. The capsule may also become inflamed from without as in acute peritonitis from any cause especially that resulting from an acute process contiguous to the liver such as perforated ulcer, acute cholecystitis, pancreatitis or appendicitis (especially one pointing upward under the liver). A cancer adjacent to or involving the liver is another cause. A retroperitoneal abscess and an acute infection of the pericardium or right pleura may also spread to the liver capsule. An acute form may be due to the gonococcus originating in gonococcal tubal infection.

### Pathology

The lesions may be localized or may involve most or all of the capsule. Exudation may occur and the exudate may be absorbed or may become plastic and the capsule may show chronic hyaline fibrous changes. It may become densely adherent to the liver, abdominal wall and neighboring structures becoming part of a generalized chronic hyperplastic peritonitis with ascites. A form of chronic adhesive perihepatitis associated with a large congested liver occurs as a part of Pick's syndrome, chronic constrictive pericarditis and is complicated by ascites with late development of cirrhosis. Many cases are parts of polyserositis.

### Symptoms

In the acute form pain over the liver, often with radiation to the epigastrium and to the tip of the right shoulder, is a fairly constant symptom. With diaphragmatic involvement there is also cough, hiccuping and aggravation of the pain with respiration and motion. In the chronic forms there may be few if any symptoms but more often there are right-sided abdominal heaviness and symptoms of ascites. In all cases the symptoms of the disease causing the perihepatitis will precede or accompany the symptoms of the perihepatitis.

### Physical Examination

The principal physical findings will usually be those of the causative disease. The liver may be enlarged and in acute cases will be tender with some muscle guarding over it. With respiration a friction rub will at times be heard over the liver. With diaphragmatic involvement respiration may be restricted and the base of the right lung may show poor aeration. Ascites constitutes a problem in diagnosis. In the chronic case peritoneoscopy may be of help.

### Laboratory Examination

The findings will be those of the causative diseases. Examination of the ascitic fluid may show specific organisms, blood or cancer cells. In Pick's syndrome the signs of cardiac tamponade associated with polyserositis and fluid in the pericardial, pleural and peritoneal cavities are diagnostic.

### X-ray Examination

X-ray films in addition to showing the findings of the causative disease may show an enlarged liver and spleen, a fixed and high diaphragm and the evidences of fixation of neighboring organs such as the gallbladder, stomach, duodenum and colon.

### Diagnosis

More or less perihepatitis may be assumed to be present as a result of an acute abdominal calamity, especially an acute peritonitis. Symptoms such as those described associated with any of the various causes are suggestive. Pick's syndrome occurs in childhood or youth, acute perihepatitis at any age and the chronic form principally in middle life. There is no single absolute diagnostic sign. The condition may not be found until operation or autopsy.

### Treatment

There is no specific treatment for perihepatitis. The treatment, medical or surgical of the cause is of course essential. Dietary care, rest, paracentesis and other measures used in any liver disease are important. No operation has been so far devised which will be of specific help.

### Prognosis

In many instances acute perihepatitis will subside with the subsidence of its causative factors. The chronic form may cause persistent symptoms, however, especially pain which may be difficult to control. As an entity, perihepatitis is not usually of great importance, but it is frequently, as in cancer, a part of a serious disease.

## Metabolic Diseases

Grouped under *metabolic diseases* are conditions resulting from metabolic disturbances including abnormal deposition in the liver of lipids, connective tissue, hemosiderin, copper, glycogen and amyloid.

### FATTY LIVER

Under fatty liver are now included the older classifications of fatty infiltration, degeneration and metamorphosis and in children idiopathic



**steatosis** It has been demonstrated that the fat deposits come not only from ingested fats but also from depots of fat storage in the body. Various mechanisms have been described to account for this deposition of fat but none has been proved to be correct. Some have assumed that the liver stores the fat to remove excess of it from other parts of the body.

### Pathology

The normal liver usually contains from 3 to 5 per cent of fat by weight. In fatty livers up to 40 or 50 per cent has been found. The liver is usually enlarged, sometimes to an enormous size and is smooth with rounded edges. The fat is deposited in large globules in and around the liver cells, with both parenchymal and Kupffer cells involved. Pressure causes atrophy of the cell substance and later interstitial fibrosis. An unhealthy liver is more liable to be affected than a healthy one. A patient with a fatty liver is less resistant to infection or poisoning than a healthy one. Many patients succumb to pneumonia and other infectious diseases.

### Etiology

Fatty infiltration may occur as a result of a variety of different causes.

1 The principal cause is *alcoholism*, not only because alcohol is a liver poison but also because with alcoholism there is usually some malnutrition.

2 *Nutritional disturbances* include not only those due to an unbalanced diet with insufficient protein and vitamins but also overindulgence in carbohydrates, fats, cholesterol and alcohol. Insufficient mastication, poor digestion due to gastric or pancreatic disorders or poor absorption due to these factors or to intestinal hypermotility may also result in malnutrition, the important cause of fatty liver.

3 A more or less severe *anoxia* is another cause, accounting for fatty liver in anemia from chronic blood loss, chronic cardiac decompensation, decreased basal metabolic rate, leukemia and rarefied atmosphere in high altitudes.

4 *Biliary stasis* may be due to chronic obstruction of the extrahepatic bile ducts or to blockage of the intrahepatic bile canaliculi.

5 *Infectious diseases*, particularly wasting diseases such as tuberculosis, cancer, streptococcal infections, typhoid, pneumonia and ulcerative colitis, are frequently accompanied by fatty liver. Focal infections are also important causes.

6 *Endocrine disturbances*, especially diabetes, thyroid, pituitary and adrenal diseases, pregnancy and shock from trauma may play a part.

### Symptoms

In uncomplicated cases there are no symptoms except those due to the causative factors. Altered metabolism may give the symptoms of

glycosuria or hypoglycemia. Reduced protein stores make the liver more susceptible to infections and toxins. At times the patient will complain that his abdomen is getting large. Serious complications due to compression are jaundice and ascites and of course hepatic coma, the last named usually found in alcoholic patients. Hepatic coma is often confused with or may occur with alcoholic narcosis or delirium tremens (see p 468).

### Physical Examination

The patient usually looks well nourished, is often fat. In the female the face is rounded and bloated, the "moon facies." The male looks eunuchoid in type and may show gynecomastia. The mouth usually shows evidences of neglect, caries, sordes and bad odor. Teeth, tonsils, sinuses and other areas may show focal infections. The skin is hot and moist, tachycardia is common.

There may be tenderness in the epigastrium and over an enlarged liver. The liver is smooth, moderately firm, with rounded edges and often cannot be felt in an obese patient. Ascites may be discernible.

Acute hepatic insufficiency will give the same findings as those seen in acute hepatitis (p 489).

### Laboratory Findings

In the blood, aside from hyperlipemia and anemia, bilirubin is commonly increased, as is urobilinogen. The *function tests* usually show normal findings except when complications have occurred. X-ray studies will show an enlarged liver. Punch biopsy will demonstrate the lesion. The principal abnormal findings are those of the causative factor.

### Diagnosis

In the presence of any of the various disease conditions known to cause it, an enlarged liver will suggest that it is a fatty liver. The liver of passive congestion can be differentiated by cardiac study, but even this type of liver may show fatty changes. The enlarged liver with amyloid and leukemic infiltration and cirrhosis is more firm. That with glycogen disease will require a biopsy for differentiation.

### Treatment

The treatment should of course consist in attempting to get rid of the cause. For the liver itself, a nutritious, well balanced diet and the other recommendations made for the treatment of hepatic insufficiency should be carried out (see p 480). Alcohol should be forbidden absolutely, permanently. Elective operations of any kind should be avoided because of the danger of the development of complications.

X ray films give an indication of the size of the liver and the presence of esophageal varices (p 207)

### Laboratory Procedures

With increasing development of the disease anemia and usually leukopenia and thrombocytopenia will be found in the blood Hypoproteinemia is common

The urine is decreased in amount and may contain much urobilinogen and some bilirubin

Liver function tests may show no abnormalities at first but later especially with the onset of portal hypertension they will help to demonstrate the degree of liver damage Reversal of the albumin globulin ratio with low albumin and high globulin occurs with ascites or preceding it Abnormal bromsulphalein retention (5 per cent or more in forty five minutes) is a common finding Elevation of serum bilirubin is relatively common as is increased cholesterol and a positive cephalin flocculation test Increased alkaline phosphatase is seen more in biliary cirrhosis Fever may occur even without complications

Fractional gastric analysis may disclose the achylia and excessive mucus of an alcoholic gastritis There may be traces of blood from surface erosion of the gastric mucosa or over esophageal varices even without actual hemorrhage

**Ascitic Fluid** In cirrhosis the fluid is a transudate as evidenced by a specific gravity of 1.005 to 1.015 The albumin content is usually low under 2.5 gm per 100 ml The fluid is usually clear but may be stained yellowish or greenish in jaundice because the bilirubin concentration is usually the same as in the blood Few cells are present mostly large endothelial type cells or leukocytes Exudates have a higher specific gravity more cells high protein and cholesterol concentration and may show blood white blood cells and malignant cells

**Esophagoscopy** will disclose the varices with overlying esophagitis often ulcerative It must be performed with caution to prevent rupture

**Peritoneoscopy** is a risky procedure but may help to clear up the diagnosis

**Punch biopsy** will usually make the diagnosis of cirrhosis but may miss areas of malignant degeneration

**Splenic blood pressure** if high may be an indication for a shunting operation (p 209)

### Diagnosis

Since over half of all cases of cirrhosis have been estimated to be due to alcoholism and since fatty liver usually precedes the cirrhosis cirrhosis may be suspected in an alcoholic patient who has had some indigestion and an enlarged liver and has continued drinking It may also

be suspected in long standing nutritional disturbances anorexia biliary stasis infectious diseases and endocrine disturbances. In such patients when the symptoms and signs of portal hypertension develop (see p 467) the diagnosis is made more probable. With increasing liver damage the liver function tests will be of value. Punch biopsy is justifiable in most cases. It is important always to determine the causative factor since treatment of this will greatly influence the course of the cirrhosis.

### Treatment

Two factors are involved in the management of cirrhosis. The cause of the disease should be treated and if possible eliminated and treatment should be instituted for the liver condition itself and its complications. Among the causes of cirrhosis avoidance of alcohol in any form is most important. Treatment of syphilis has a good effect. Establishment of cardiac compensation relief of external biliary stasis and adequate treatment of infection and endocrine disease are all of value. Thorough eradication of all focal infections is important. Treatment of the liver involvement and its complications has been discussed under treatment of liver diseases in general (p 480). Primary cancer is an irremediable complication. Operations of any kind except for ascites or varices should be avoided if possible but if they are absolutely necessary the patient should be treated adequately before and after operation to avoid liver failure and coma (see p 484).

### Prognosis

Though cirrhosis was formerly considered to be invariably a fatal disease there are now many patients who when adequately treated will be restored to normal or will at least have sufficient functioning liver tissue to permit of many years of almost normal existence provided all causes have been permanently eliminated.

### HEMOCHROMATOSIS

Essentially a disease in which the pigments hemosiderin and hemofuscin stain the skin and many viscera. hemochromatosis is usually discussed under hepatic cirrhosis (p 506) because these pigments have their most marked effect on the liver. Practically all tissues show the pigmentation even the brain and the endocrine glands being stained. Some degeneration and fibrosis develop as a result. The liver becomes cirrhotic the pancreas fibrotic and the gonads show atrophy of the germinal epithelium. The reason why the pigments derived from the blood become so widely distributed in the tissues is not known. Various explanations have been advanced none proved. Hemochromatosis appears to be due to a congenital error of metabolism as a result of which the iron absorbed into the cells does not get excreted from them. It may

be brought on by excessive transfusions *Hemosiderosis* with which it may be confused usually follows multiple transfusions or may be associated with malnutrition excessive ferrous therapy or some forms of severe anemia. However the iron deposition in the tissue does not produce the degenerative changes observed in hemochromatosis. It may also be confused with the rare disease known as *black liver* which is accompanied by chronic jaundice variable liver function tests nonfunctioning gallbladder and at times melanuria. The black pigment which stains the liver has not been identified. The course of black liver is benign it is recognized by biopsy or at operation.

Hemochromatosis occurs almost exclusively in men and more than half of the cases occur in the fourth to the sixth decades of life. It occurs all over the world is occasionally definitely hereditary and may be associated with a severe refractory anemia.

### Symptoms and Signs

The *classical findings* consist of an enlarged liver diabetes mellitus skin pigmentation impotence and loss of body hair. Before the advent of insulin most patients died of diabetic coma and rarely survived more than a few years. Today life may be prolonged for many years although most patients will die in three to five years principally from hepatic insufficiency congestive heart failure bleeding esophageal varices bronchopneumonia or malignant hepatoma. Other causes of death include various complications occurring in other affected organs.

*Symptoms* usually start insidiously mild cases may go on for years before definite symptoms occur. In general the symptoms consist in weakness lassitude dyspnea and weight loss. Some patients will complain of retrostaltic gastrointestinal symptoms or a feeling of weight due to the enlarged liver. Others may be troubled about the pigmentation the impotence or marked loss of hair. Many will not reach a doctor until complications develop. These may be manifested by hematemesis or ascites from cirrhosis pernicious anemia from gastric gland atrophy endocrine disturbances such as diabetes adrenal insufficiency hypothyroidism or hypogonadism heart or renal failure pneumonia or cancer of the liver.

### Diagnosis

The diagnosis is not difficult when the classical findings mentioned previously are all present. This is not always the case however Hemochromatosis should be suspected when a patient with diabetes does not respond normally to insulin and shows skin pigmentation of the typical bronze or slate blue or gray color. The excessive weakness and loss of weight in spite of good medical care are also suggestive and may be due to endocrine changes. Careful inquiry about the symptoms of the compli-

cations may help to clinch the diagnosis. When hematemesis occurs it may be due to bleeding varices or vitamin deficiency or may originate in a cancer involving the stomach. The anemia should be studied carefully. If typical of pernicious anemia it may be due to the degeneration of gastric glands caused by iron deposition. The heart, lungs and kidneys should be studied regularly for evidence of changes secondary to the disease.

### Laboratory Findings

These are not particularly helpful. The blood will usually show some anemia and hyperglycemia until complications change the picture.

*Urine examination* aside from glycosuria may at times show granules of hemosiderin in the shed epithelial cells. Complications will be manifested by the presence of bilirubin, porphyrins, acetone, albumin or casts.

*Gastric analysis* if considerable gland atrophy is present will show a persistent anacidity and blood will be found from varices or from complicating cancer.

*Duodenal contents* if the exocrine function of the pancreas is affected, will show evidence of deficiency of pancreatic ferments and the stools may show undigested meat fibers, starch granules and neutral fat.

*Tests* Most of the tests formerly recommended are of little if any specific value. The *Fishback intradermal test* has not been found reliable. *Radio iron* absorption studies have been inconclusive. *Biopsy* of the liver either directly or by means of peritoneoscopy is most significant; biopsy of the skin, not so good. Results of *liver function tests* will depend on the amount of liver damage and will correspond to those found in cirrhosis from other causes. Other complications will show their characteristic findings.

### Differential Diagnosis

This will include differentiation from simple cirrhosis, uncomplicated diabetes, uncomplicated liver cancer, primary pernicious anemia, Addison's disease or other endocrine disturbances. The pigmentation of *pellagra* and its attendant gastric anacidity and of *argyria* with its history of silver medication may be fairly easy. Hemosiderosis found on pathologic study does not present the classical symptoms of hemochromatosis and most frequently follows repeated blood transfusions.

### Treatment

Until recently the treatment consisted largely in conservation of liver function by means of a diet suitable for any hepatic lesion, as described before (p. 491). Complications such as diabetes, endocrine disturbances, pernicious anemia and others also mentioned should be appropriately treated. Antibiotics may be used for intercurrent infections.

*Specific therapy* involving massive phlebotomy with the removal of 500 to 1000 ml of blood each week over an indefinite period has shown good results. If hemoglobin drops below 10 gm per 100 ml the treatment is suspended until it is restored to nearly normal. For this transfusions may be indicated.

The use of chelating agents to remove iron from the body through the urine has been tried extensively but is not as efficacious as repeated phlebotomy.

Patients with refractory or pernicious anemia may require repeated transfusions in addition to the usual therapy with liver extract, folic acid or vitamin B<sub>12</sub>.

### Prognosis

Before the advent of these newer treatments the average duration of life formerly from a few months to a few years had been prolonged to five or more years by adequate treatment of diabetes. The newer methods may extend even double this life expectancy although it must be realized that many other changes that have occurred in the liver, pancreas and elsewhere are not compatible with normal life expectancy.

### HEPATO-LENTICULAR DEGENERATION

#### (WILSON'S DISEASE)

Hepatolenticular degeneration is a rare disturbance of copper metabolism characterized by the development of cirrhosis with degeneration of the lenticular nuclei due to deposition of copper. Its *symptoms* include fever, vomiting, diarrhea followed by symptoms of portal hypertension with enlarged liver and spleen, jaundice and epistaxis. In the early stage a narrow brownish green ring of copper may be seen at the outer rim of the cornea. After an interval of apparent subsidence of symptoms signs of cerebral injury from the deposition of copper may occur including athetoid movements, spasms, contractions, hyperreflexia and deglutition disturbances. The disease was formerly considered invariably fatal but some results have been obtained by attempting to rid the tissues of copper by the use of BAL, the anti Lewisite drug, and the usual treatment for cirrhosis (p. 509).

### THE LIPIDOSES

#### (XANTHOCHROMIATOSES)

With the finding of an enlarged liver the clinician must bear in mind the fact that some rare familial disorders of lipid metabolism often grouped under the terms *xanthochromatosis* or *lipoid granulomatosis* may account for this enlargement. Essentially these conditions are due to disease of the reticuloendothelial cells and histiocytes caused by an intracellular disorder of cholesterol metabolism. They may or may not be

associated with hyperlipemia and hypercholesterolemia. Although these are usually childhood diseases, occasionally a case may occur in the adult. A striking feature is the presence of yellowish xanthomatous deposits in the skin, especially of the eyelids, fingers and elbows, which may attract attention before the deposits are found elsewhere in the body. However, they are not invariably present in the skin. Deposited in the liver, they may cause simple enlargement, may cause pressure on the ducts, producing biliary cirrhosis, or may occur as nests in the liver, spleen and lymph nodes. Splenomegaly is the rule.

Included in this group are such diseases as *Gaucher's disease*, in which the cerebroside *kerasin*, normally occurring only in the brain, is found in the so-called Gaucher foam cells in the liver, spleen and various other parts of the body. These cells may be identified in tissue obtained by sternal or splenic puncture.

*Niemann Pick disease* is a similar condition, in which the cells are filled with a phosphatide, *syringomyelin*. This also causes marked enlargement of the liver and spleen. The children rarely live more than three years.

*Hand Schüller Christian disease* belongs in the group, but in this condition cholesterol and esters are deposited in the form of nodules in the periosteum of bones and in the dura mater. Involvement of the pituitary body may cause diabetes insipidus or pituitary dwarfism. The liver and spleen are not markedly involved.

In diseases associated with prolonged *hyperlipemia*, such as diabetes or severe acute hepatitis, xanthochromatous accumulations may occur in the liver, spleen and other organs, and their appearance in the skin is often marked, but usually not before jaundice has developed.

### Treatment

For all these conditions there is no specific treatment. The liver lesions must be treated in the same way as any hepatic insufficiency, and the specific disease with which they are associated must be cared for.

### Prognosis

Children afflicted with these diseases rarely live more than a few years. Most adults with *Gaucher's disease* succumb to intercurrent diseases within a few years.

## GLYCOGEN DISEASE (VON GIERKE'S DISEASE)

In glycogen disease there is an abnormal accumulation of glycogen, mostly in the liver and kidneys. It is a familial disease, starting at birth or early life. The principal findings include hepatomegaly due to the deposits of glycogen, hypoglycemia, acidosis, impaired blood sugar response to epinephrine, increased sensitivity to insulin, hyperlipemia and



hypercholesterolemia. The liver is large, smooth, firm, like rubber and pale yellow and may show enormous deposits of glycogen with considerable deposits of fat distending the parenchymal cells but not the Kupffer cells. Some glycogen is also deposited in the kidneys, head, brain and spinal cord.

### Etiology

The disease is considered to be due to a defect of glycogenolysis resulting in accumulations of glycogen in the tissues. It is supposed to be influenced by enzymes, hormones and the autonomic nervous system. It is definitely not due to hyperinsulinism or adrenal defect.

### Symptoms

In early childhood an enlarged abdomen, fat face, underdeveloped extremities, clumsiness and easy fatigability call attention to this disease. The hypoglycemia may induce convulsions, often diagnosed as epilepsy. When ketosis supervenes, vomiting may be the first symptom. There are no other physical findings than the enlarged liver, since portal hypertension does not occur.

### Laboratory Findings

The findings in the blood as mentioned before, though fairly constant, are not characteristic. The liver function tests usually show normal findings. Serum protein and enzymes are not affected. There may be a mild anemia.

### Diagnosis

Liver biopsy is the only method of differentiating this disease from fatty liver. Other types of hepatomegaly are mentioned in the general section on the liver (p. 470).

### Treatment

There is no specific treatment. The usual measures used in liver disease should be carried out, but in practically all cases the children die in infancy and childhood, frequently of intercurrent disease.

### PORPHYRIA

Porphyria is a rare dyscrasia in which porphyrin is found in the urine and feces. Porphyrin, an iron-free hematin, is believed not to be derived from a breakdown of hemoglobin but to originate in ingested animal and vegetable tissues. The exact cause of this abnormal metabolism is not known, but as the liver normally is concerned with porphyrin metabolism, it has been assumed that porphyria is due to a liver disturbance. It has also been suggested that zinc ions may cause the enzymatic block thought to be responsible for this dyscrasia. A number of drugs are known to cause

it including excessive use of barbiturates sulfonamides alcohol arsenic and heavy metals especially lead and selenium Occasionally porphyria is associated also with hemolytic jaundice liver necrosis miliary tuberculosis pellagra and some bacterial and mycotic diseases Porphyrins have been known to cause degenerative changes in various tissues including the nervous system liver and kidneys These have been attributed to a relative ischemia due to vascular spasm

### Symptoms

There may be no symptoms other than the peculiar red wine color of the urine noticed by the patient with no history of the ingestion of food or drugs known to produce this color When symptoms are present they may be mild or severe the most constant being photosensitivity often of such a degree that exposure to sunlight may induce violent attacks with abdominal and central nervous manifestations in varying degree

### Types

Three types of porphyria have been described

1 The *congenital type* occurring mainly in male infants and frequently fatal

2 The *acute type* usually seen in middle aged women with symptoms resembling those of an "acute abdomen" the patient being subjected occasionally to emergency operation with no lesion found on careful exploration The symptoms consist in severe colicky abdominal pains with nausea vomiting and constipation often combined with evidence of cerebral irritation and peripheral neuropathy suggesting intracranial or spinal cord tumor

3 The *chronic type* occurs in older women and presents some or all of the symptoms of the acute type but in mild form Diabetes evidences of hepatic disease jaundice and hyperpigmentation may be found Acute exacerbations may occur

### Physical Examination

The abdominal findings are not commensurate with the severity of the symptoms Even in the acute case tenderness and muscular rigidity may be mild or absent Distention may be mild x-ray studies showing only moderate amounts of intestinal gas

### Laboratory Tests

Some degree of sodium and chloride deficiency is revealed but the only specific finding is the redness of the urine shown by chemical or spectroscopic tests to be due to porphyrin

### Treatment

No specific treatment has been found although good results have

hypercholesterolemia The liver is large smooth firm like rubber and pale yellow and may show enormous deposits of glycogen with considerable deposits of fat distending the parenchymal cells but not the Kupffer cells Some glycogen is also deposited in the kidneys heart brain and spinal cord

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Great sensitivity to insulin may at times be accompanied by excessive dextrose excretion with fatty liver. Lipocur, the pancreatic fat "hormone," has been found to relieve this condition. "Lipocur deficiency" therefore has been mentioned as a cause.

Other liver diseases may of course complicate diabetes and are of serious import. They must be treated in the usual way, but control of the diabetes must be carefully regulated, always avoiding hypoglycemia.

### THE LIVER IN THYROID DISEASE

Because of its effect on metabolism and its relation to other endocrine glands, the thyroid exerts a definite influence on the liver. Thyroid disease tends to produce fatty changes in the liver.

With a severe *thyrotoxicosis* there is a tendency for acute parenchymal damage to develop, even to the point of acute yellow atrophy. The liver is further damaged by the congestion due to cardiac decompensation as a result of hyperthyroidism and by nutritional deficiency. Long continued thyrotoxic states may produce chronic hepatitis or portal cirrhosis. In thyroid disease, liver damage should be searched for even before the development of actual hepatic symptoms. Jaundice is a sign of serious import, being often associated with acute yellow atrophy.

Liver function tests will usually show the results of the excessive metabolism. Blood sugar, protein, especially albumin, and cholesterol values are low. Other tests will vary according to the degree of liver damage and may be affected by the concomitant cardiac damage. In any case of thyrotoxicosis it is important that liver function tests be performed and important also to preserve hepatic function by means of the regimen recommended for hepatic insufficiency, including diet, vitamins and other measures (see p. 480). In acute parenchymatous disease the treatment recommended for this condition should be carried out in conjunction with adequate treatment for the thyroid condition. Infections must be avoided and if present treated vigorously, since these patients show poor resistance.

In *hypothyroidism*, whether primary or produced by thyroidectomy, an increase in blood cholesterol is the rule. Serum amylase and alkaline phosphatase may be low, and a hypoglycemia is commonly found. Urinary calcium is low. The liver may become fatty. The degree of cholesterol concentration in children has been used as a guide to thyroid dosage.

### Neoplasms of the Liver

#### BENIGN NEOPLASMS

#### BENIGN TUMORS AND CYSTS

Benign tumors of the liver are rare. Angiomas, usually hemangiomas, are the most frequent. Adenomas, generally fibroadenomas, are less com-

been reported from the use of chelating agents such as BAL (British anti Lewisite) in the acute cases

*Prophylaxis* consists in avoidance of overexposure to sunlight of barbiturates alcohol and the other drugs known to cause the disease

### AMYLOID DISEASE OF THE LIVER

Amyloidosis in general is rare although in chronic disease hospitals especially those for the treatment of tuberculosis it is a frequent complication It is most common in chronic pulmonary tuberculosis long untreated syphilis and chronic suppuration It is also seen in rheumatoid arthritis and heart disease leukemia cancer and prolonged malnutrition A secondary infection will frequently be the exciting cause

### Pathology

Amyloid a protein of cartilaginous hardness is deposited in small arteries and capillaries and interlobular lymph spaces This causes uniform enlargement of the liver which is firm and feels rubbery Since bile channels are not involved there is no jaundice and portal hypertension is unusual There is no interference with liver function Cirrhosis may be a late complication

### Diagnosis

Diagnosis is made by biopsy or at autopsy or by the Congo red test carried out by injecting intravenously 10 cc of a 1 per cent solution of Congo red and testing for it in the blood in one hour Normally over 75 per cent of the dye is still present but in amyloid disease it is absorbed by the amyloid substance so that little remains

### Treatment

The treatment for the original causative condition is most important The liver function can be conserved by the measures used in hepatic insufficiency and care can be taken of hypoproteinemia There is no way of getting rid of the amyloid but occasionally patients cured of the original condition may be relieved of hepatic symptoms The presence of amyloid deposits does not alter the prognosis of the original disease

### THE LIVER IN DIABETES

In diabetes an enlarged liver is rather frequent in children but occurs rarely in elderly adults The fatty liver due to malnutrition occurring in uncontrolled diabetes usually clears up under insulin therapy and adequate diet At times there may be hyperlipemia rarely coming not controlled by insulin As a rule weight reduction is most beneficial

A liver enlargement may be caused at times by a rapid accumulation of water enough to distend the capsule and to produce pain

Great sensitivity to insulin may at times be accompanied by excessive dextrose excretion with fatty liver. Lipocue, the pancreatic fat "hormone" has been found to relieve this condition. "Lipocue deficiency" therefore has been mentioned as a cause.

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Benign tumors of the liver are rare. Angiomas, usually hemangiomas, are the most frequent. Adenomas, generally fibroadenomas, are less com-

mon and other tumors such as hamartoma leiomyoma and teratoma are curiosities. Tumors may occur in any part of the liver. Their exact etiology is not known but it has been supposed that regeneration of liver tissue as a result of a small area of injury, irritation or necrosis may cause an overgrowth in the area producing a tumor. Any of them may undergo malignant changes or rarely may become infected.

*Cysts* may be of parasitic (hydatid) or nonparasitic types. Nonparasitic types may be retention cysts due to blockage of tiny liver ducts or to cystic changes in benign tumors or they may be endothelial in origin. The multiple cysts of varying size occurring with polycystic disease of the kidneys, spleen, pancreas and elsewhere are rare. Nonparasitic cysts may occur at any age but most of them are found in the fourth and fifth decades and primarily in men.

*Hydatid cysts* are produced by swallowing eggs of the *Taenia echinococcus granulosus*, a tiny tapeworm living in the intestinal tracts of the dog, cat and wolf. They are common in Italy. They may be found in water or food contaminated by these animals or on the hands of children playing with infested pets. The cysts are usually unilocular and are filled with small daughter cysts. They are found mostly in adults, more frequently in females. They grow slowly, those starting in childhood frequently do not grow to large size until later in life. All but a few occur in the right lobe. They tend to rupture when they are large. Alveolar hydatid cysts are rare. They act like malignant growths, infiltrating the liver and metastasizing by external sprouting. They are usually adherent to neighboring structures and are associated with splenomegaly. The *Taenia echinococcus* is discussed in the chapter on intestinal parasites (p. 161).

### Symptomatology

Practically all tumors and cysts are symptomless until they grow to a large size or develop complications. Some may become so large that a patient feels a sense of weight in the abdomen or can palpate a tumor which may move from side to side with changes in position. Increased intra-abdominal pressure upward against the diaphragm may cause dyspnea. With pressure upon or involvement of nerves, constant or intermittent pain may occur in the abdomen or back or be referred to the chest. With encroachment on neighboring organs there may be retrostaltic symptoms, nausea, vomiting, anorexia, constipation and even biliary colic. Associated cirrhosis will give the symptoms of this disease with hepatomegaly, splenomegaly, esophageal varices, jaundice or ascites. Rupture of a cyst or twisting of its pedicle will cause sudden pain. If the rupture is into the peritoneal cavity, signs of peritonitis will occur; if into the pleura or lung, evidences of pleuritis or pneumonitis; if into the biliary tract, jaundice and pain. Secondary infection may occur in any cysts and produce an abscess which will cause fever and tenderness. Rupture of a hemangioma will result in a hemoperitoneum.

### Physical Examination

Uncomplicated early tumors and cysts will rarely show any abnormalities on physical examination. When they first become palpable they will be small and not differentiable. As they grow larger their consistency may give a hint as to the type. Hemangiomas may be compressible and on auscultation a hum or murmur may be heard over them. Some have been described as extending almost to the pubes. Large cysts may show a fluid wave, hydatids a thrill or vibration. Solid tumors will be firm. The liver as determined by palpation and percussion may be enlarged. Tenderness is not characteristic and usually occurs with the complications mentioned above. With cirrhosis the usual findings in this disease will often mask the presence of a tumor or cyst. The finding of enlarged polycystic kidneys and other masses suggests a similar process in the liver.

### Laboratory Findings

For tumors and nonparasitic cysts no characteristic findings are present until complications develop. If liver function tests show impairment they will usually indicate a large tumor causing pressure necrosis, a secondary hepatitis due to infection, or a marked cirrhosis. Although a mild leukocytosis is occasionally present if marked it would indicate a secondary infection, perforation or intercurrent disease, and fever would also be present. Anemia due to malnutrition in patients with anorexia is not characteristic. Urines are negative except for albuminuria in polycystic disease. Stools would be negative except with complications especially hemorrhage.

In the case of *hydatid* cysts specific immunologic tests are available but none are as valuable as the simple skin test with antigen prepared from heterologous tapeworms. This test has been shown to be diagnostic in over 90 per cent of cases. It is read in ten to thirty minutes, fading out in one hour. Since these cysts have a tendency to rupture when they get large the peritoneal and pleural fluid may give their characteristic findings. "Daughter cysts" or fragments of membrane may appear in the stools. Eosinophilia, often of high degree, varying from 20 up to 70 per cent of the total white cell count, is a constant and helpful finding although it usually disappears if the cyst becomes calcified.

### X-ray Studies

X-rays are not of much help. When the tumors are too small to be palpable liver visualization by means of Thorotrast or by splenoportography may at times disclose localized areas of increased or decreased density. Calcified tumors or cyst walls will be seen occasionally on scout films (Fig. 93). As mentioned before, x-rays are not as reliable as palpation in estimating the size of the liver or spleen, but with a barium





Figure 93 Hydatid (Echinococcus) cyst of liver. Note the partial calcification of the periphery and the uneven shadows of the interior, some suggesting ringlike shadows of daughter cysts.

meal and enema and cholecystography. Evidence or displacement of the stomach, intestines, and gallbladder by an enlarged liver or by a tumor or cyst is of distinct value. The finding of esophageal varices would be of help. With pneumoperitoneum the size and conformation of the liver and of masses can be made out.

### Biopsy

Punch biopsy is uncertain. Blindly, there is no certainty that the needle will enter the tumor. Diagnosis of a normal liver or cirrhosis might be made even though a tumor or cyst were present. There is great danger in puncturing a hydatid cyst or an infected cyst or tumor, since a spread might be caused by withdrawal of the trocar.

Peritoneoscopy may at times be used to visualize the mass. A cyst could probably be recognized; the nature of a tumor might be observed and biopsy under direct visualization would be a fairly safe procedure except in the case of hydatid cyst.

### Diagnosis

The diagnosis is usually difficult and in the absence of early symptoms and physical signs is generally not determined until late. Upon palpation of a mass, differentiation among hepatic neoplasm, enlarged gallbladder

and gastric pancreatic or colonic tumor must be made. The finding of impaired liver function may be present in a severe hepatitis an advanced cirrhosis often with carcinoma or a large tumor or cyst. The history may be of little aid although being of Italian nationality is a helpful factor in hydatids. Except in the case of hydatid cyst no characteristic laboratory findings occur until complications produce their own manifestations. The occurrence of a tumor without other symptoms or signs should arouse suspicion of a benign lesion. As stated before an x-ray study may be of assistance and a biopsy preferably by means of peritoneoscopy may clinch the diagnosis.

### Treatment of Hepatic Tumors

There is no treatment for any hepatic tumor except surgical removal. X-ray therapy has a destructive effect on the liver parenchyma even when it is given for cancer elsewhere so that its use for liver disease is definitely contraindicated. Chemotherapy is of no value. Retrostaltic symptoms and anemia can be controlled by an adequate well balanced diet and other procedures as recommended for liver disease at the beginning of this chapter (p. 480).

Surgical excision is now being done by skillful surgeons with comparative safety and good results. The operative mortality rate should be under 10 per cent. Because of the danger of complications especially malignant degeneration operation is the best procedure except when definite contraindications are present. Even poor risks may be converted into good ones by adequate diet therapy.

### Treatment of Hepatic Cysts

There is no medical treatment for cysts. Fluid aspiration is dangerous especially in hydatid cysts because of the danger of spread.

The only treatment of cysts is surgical. Small pedunculated cysts should be excised completely. Larger noninfected cysts may be enucleated and the capsule sterilized with formaldehyde and closed. On occasion a protruding hydatid cyst may be delivered through the wound without the necessity of suture or drainage. Infected cysts or large uninfected hydatid cysts are usually treated by aspiration of contents followed by injection of 0.5 to 2.0 ounces of 10 per cent formaldehyde solution. The formalin is removed in five minutes the cyst cavity incised the interior is sterilized with formalin and external drainage or marsupialization instituted. Internal drainage by anastomosis between the nonparasitic cyst and the small intestine is of value. Polycystic livers are of course inoperable as are alveolar cysts.

### Prognosis

After an operative mortality rate of 10 to 20 per cent the ultimate prognosis depends on the nature of the lesion. Hemangioma is the

most favorable tumor having both low operative mortality rate and a good prospect of cure. In tumors showing malignant degeneration the prospect is poor. External drainage from cysts may last for years after operation. Hydatid cysts are apt to recur in nearly half of cases because of the spilling of contents at aspiration or operation. With alveolar cysts the patient may live up to eight years.

### HEPATIC GRANULOMAS

The use of liver biopsy has called attention to the fact that nonspecific granulomas of various sizes resembling tubercules are frequently found in the liver associated with various diseases. They have appeared in patients with sarcoidosis, tuberculosis, syphilis, erythema nodosum, brucellosis, tularemia, infectious mononucleosis, histoplasmosis, actinomycosis, schistosomiasis and lymphogranuloma venereum. They are usually composed of thin connective tissue containing mononuclear cells and occasional foreign body giant cells. Liver tissue surrounding the granuloma may show the specific findings and cultures may reveal the organisms of the disease with which the granulomas are associated. So far as is known they cause no symptoms but may initiate later cirrhotic changes in the liver. When granulomas are found a complete survey of the patient is indicated to find the disease with which the lesions are associated. Diseases involving the gastrointestinal tract are discussed under their appropriate titles. No specific treatment for granulomas is required.

### SARCOIDOSIS

A disease of unknown origin in which granulomatous lesions appear in various parts of the body, sarcoidosis may rarely involve the liver. It can be diagnosed only by biopsy, usually of the skin lesions. It may be assumed to be the cause of liver tumors if sarcoid granulomas have been found elsewhere, especially in the skin, parotid glands and eyes. It is frequently associated with tuberculosis. There is no specific treatment for this disease but it may clear up during treatment for tuberculosis or disappear spontaneously. Corticosteroids have been tried but with no definite success. The liver should be treated as for cirrhosis (p. 509). This condition is rarely fatal.

### MALIGNANT NEOPLASMS

Cancer of the liver is common since many cancers in the abdomen, pelvis, chest and elsewhere metastasize to the liver. Primary carcinoma is rare, only about one sixtieth as frequent as the metastatic form. Primary sarcoma is really a freak when it occurs and even secondary involvement with sarcoma or Hodgkin's disease is rare. Leukemia produces definite liver changes. Since even a small fraction of normal liver tissue can carry

on liver function malignant involvement is frequently overlooked especially the primary type which usually occurs with cirrhosis

### Pathology

*Primary carcinoma* (malignant hepatoma) of the liver is of three types the hepatocellular type originating in the polygonal cells usually called hepatoma the cholangiocellular type originating in the small bile ducts called cholangioma and a mixed type containing both elements Although probably unicentric at first it is usually multiple when first seen producing diffuse nodules like hobnails which are not umbilicated as are those of secondary cancer At times there is one large massive tumor which may involve the whole right lobe with many smaller ones in the left lobe Cancerous thrombi are usually found in portal and hepatic venules accounting for the diffuse spread which occurs at first intrahepatically to produce the many nodules and later spreads in about half of the cases to the gallbladder pancreas bones heart and lungs

*Secondary carcinoma* occurs as multiple yellowish white or grayish green hard nodules varying in size and coalescing to form larger masses The primary lesion may be almost anywhere in the body the most frequent locations being the gastrointestinal tract pancreas lung breast pelvis or thyroid

*Lymphomas Hodgkins disease* involves the liver in nearly half of all cases of this disease It may cause some enlargement of the liver due partly to infiltration with Hodgkins disease partly to fatty changes On the surface there may be nodules resembling secondary carcinoma or leukemic infiltration A slightly enlarged liver may be overlooked As the infiltration continues liver functions gradually become impaired and jaundice may supervene caused in part by the infiltration partly by hemolysis Chylous ascites is a rare complication The spleen is usually enlarged Biopsy either by the punch method or at operation is often necessary to establish the diagnosis unless superficial glands showing the lesions make it probable that the enlarged liver is involved

*Sarcomas* look like cancer and are found in large cirrhotic livers one third occurring in males at the two extremes of life

*Other Malignant Tumors* Malignant teratoma melanoma chorio epithelioma and hypernephroma are occasionally encountered Myelomatosis may be accompanied by accumulation of atypical plasma cells in the sinusoids of the liver

*Leukemia* The liver is usually enlarged in acute and chronic leukemia but particularly so in the chronic lymphatic form owing to leukemic infiltration of the acini In the myelogenous form it is mostly the capillaries that are involved Enlargement of the liver and spleen may resemble that in Hodgkins disease The symptoms of leukemia with acute exacer

bitions may be complicated by those of hepatic insufficiency. Jaundice may be due to hemolysis or to pressure of enlarged glands. Ascites is an occasional complication. The treatment is the same as for leukemia with added attention to hepatic insufficiency.

*Leukemoid reactions* with white blood cells up to 100 000 or 150 000 may rarely occur in primary carcinoma of the liver with metastases. Fever is a common finding in such cases.

### *Etiology*

Various conditions predispose to cancer. Cirrhosis is most frequent. Hemochromatosis and cholangitis are also causes; the last named preceding cholangioma. Parasites invading the liver and syphilis by causing cirrhosis may also be precursors. The ingestion of carcinogens such as food dyes especially azo compounds has been suggested as a cause. Dietary deficiencies especially of the B vitamins may predispose to cancer since riboflavin protects the organism from the effects of azo compounds as does avidin, a constituent of egg white. Congenital defects, teratoma and adrenal rests may become malignant. Passive congestion, trauma and probably heredity must be included.

### *Symptoms*

Patients with previous cirrhosis, the most common precursor of cancer, are often treated for simple cirrhosis without suspicion of malignant disease. The diagnosis is frequently made only at operation or autopsy. Some patients may have symptoms of an abdominal calamity and be operated on for this condition; some with fever may show symptoms suggesting hepatic abscess. Patients with no previous gastrointestinal symptoms may have few complaints until liver failure calls attention to an enlarged liver. Others may go on to massive involvement and metastases before recognition.

### PRIMARY CARCINOMA

*Primary carcinoma* usually has an insidious onset with rapid development; patient rarely living as long as six months from the onset of symptoms. Early manifestations may consist of indefinite retrostaltic symptoms, anorexia, weight loss and asthenia. Pain in the epigastrium, right upper quadrant or back, if pain is present at all, usually increases with the size of the growth. Hypoglycemic symptoms are frequently present. Jaundice is not constant and may often appear late. Pruritus, frequently severe, may occur before jaundice becomes evident. The patient may die before jaundice develops. Hemorrhages from the nose, from varices in the esophagus and rectum and from ecchymoses and purpuric areas may occur fairly early. Ascites is a later symptom. Lung metastases may cause pre-

dominant symptoms. Hepatic insufficiency with delirium, somnolence, stupor and coma completes the picture.

### Examination

General findings of cachexia are usually present. The liver is enlarged and hard, and in one third of the cases there is enlargement also of the spleen as a result of cirrhosis and its portal hypertension. At times a large single tumor or multiple tumors may be felt. Jaundice occurs late if at all as does ascites with the fluid blood stained or actually bloody. Fever is usually present and moderate but at times is high and spiking suggesting abscess. Progressive decrease in blood pressure and lowered blood sodium together with great weakness suggest adrenal involvement.

### Laboratory Examinations

The blood will show anemia, occasional high leukocytosis and increased sedimentation rate according to the degree of necrosis. Leukemoid reactions with counts over 100,000 have been found with extensive metastases. Occasionally hypoglycemia may be present. Icterus occurs in about one half of cases but serum bilirubin is generally not high. Serum alkaline phosphatase is usually elevated. Liver function tests as a rule are not affected except by cirrhosis or duct obstruction as in Hodgkins disease. Stools may be light, bulky and fatty. Urine will contain bile and albumin and may contain bilirubin before jaundice occurs.

### X-ray Examination

X-ray studies may disclose an enlarged liver but this is deceptive. A large spleen may also be seen. Visualization of the liver by injection of Thorotrast may show areas of decreased density representing the tumors. Angiography can also be used to show tumors. Chest x-rays will reveal mediastinal and lung metastases.

### Peritoneoscopy

In doubtful cases peritoneoscopy may decide the diagnosis when a tumor can be seen and biopsy taken.

### Punch Biopsy

If the specimen happens to be taken from a tumor nodule, biopsy will be of distinct help but in some cases when repeated punch biopsies have been done and no tumor tissue found, an erroneous diagnosis of cirrhosis may be made.

### Differential Diagnosis

This is usually difficult. Primary cancer should be suspected in any

bations may be complicated by those of hepatic insufficiency. Jaundice may be due to hemolysis or to pressure of enlarged glands. Ascites is an occasional complication. The treatment is the same as for leukemia with added attention to hepatic insufficiency.

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The symptoms are those of the infectious diseases with added symptoms due to the liver involvement all varying somewhat according to the different diseases and the hepatic lesions they induce. Frequently the symptoms of portal hypertension are the first to call attention to the liver.

On physical examination there is usually more or less enlargement or tenderness of the liver and when portal hypertension is produced the symptoms are those of this condition regardless of the cause.

**Laboratory Findings** In some cases liver function tests may aid in making the diagnosis in others they may show no abnormal findings until late. *Biopsy* if there is no contraindication to it may be required to make the diagnosis.

The treatment in each case will of course be that of the infectious disease plus adequate care of the liver by diet vitamins and other measures as recommended for liver disease in general.

Some of the infectious diseases affecting the liver particularly are as follows

#### WEIL'S DISEASE OR SPIROCHETAL JAUNDICE

Weil's disease is caused by *Leptospira icterohaemorrhagiae*. It is really a systemic infection its toxins particularly affecting the capillaries the liver spleen kidneys adrenals pancreas the gastrointestinal tract the muscles and other organs. Capillary degeneration is the cause of the hemorrhagic features of the disease with bleeding from all mucous membranes and into the skin under serous membranes and the meninges. Skeletal muscles especially the gastrocnemius deltoid and pectoral muscles may show degenerative changes. The liver involvement may vary from edema and cloudy swelling with vacuolization of parenchymal cells to actual degeneration and necrosis resembling severe infectious hepatitis or acute yellow atrophy. The accompanying icterus stains all tissues. The kidneys usually are badly damaged and all tissues of the body are injured in varying degrees both by the toxins of the spirochete and by the jaundice. Involvement of the pancreas will resemble acute pancreatitis.

#### Etiology

The disease is most prevalent in warm climates is rarely seen in the north and then usually in the summer months. The organism is one of a group of spirochetes causing febrile diseases in various parts of the world. These spirochetes are found in the urinary and intestinal tracts of many animals including mice rats foxes and domestic animals and can live for weeks in stagnant water. Consequently infection occurs most frequently in sewer workers fishermen fish handlers butchers farmers and other field workers and in miners. The organisms enter the



case of cirrhosis with the development of a large and often painful liver and ascites. The cirrhotic liver with ascites is usually small. Gumma can often be ruled out by finding positive serologic evidence of syphilis and by the therapeutic test, but even if present it may be associated with cancer. Multiple nodular hyperplasia is not usually accompanied by a large liver.

#### SECONDARY CARCINOMA

*Secondary cancer* takes a longer course than primary, is frequently associated with cirrhosis, usually shows no secondary splenomegaly, and at operation the tumors are seen to be umbilicated. Furthermore, the primary lesion elsewhere, which has metastasized to the liver, can usually be located.

#### Treatment of Hepatic Cancer

As in other parts of the body, surgical removal of cancer offers the only hope of cure.

With modern operative technique, *primary cancer*, if not too diffuse, can be resected, but fewer than 25 per cent of such cases are considered operable. These operations are often performed under hypothermia. Even as much as a lobe of the liver has been successfully removed. The remaining liver tissue will usually undergo sufficient hyperplasia to take over liver function. Unfortunately, most such operations can be classed only as palliative, since recurrences are common, death occurring usually within four months.

*Metastatic hepatic cancer* cannot be removed, except when by chance a few discrete nodules, excised when found at operation for the primary growth, turn out to be the only metastases.

*Medical care* is palliative and should consist in a diet such as is used in any liver disease (p. 481), analgesics and sedatives to keep the patient comfortable, and repeated paracenteses to relieve the discomfort of the much distended heavy abdomen. Mercurial diuretics may be tried, but the effect is usually disappointing.

*Radiotherapy* may be of value in treating radiosensitive lymphomas, but cannot be classed as really curative. The use of x-ray to combat the pruritus is also usually of little or no value. In Hodgkin's disease, radiotherapy and chemotherapy are used as in cases of this disease in general. In carcinoma, no successful form of treatment except surgery has been suggested.

### General Diseases Affecting the Liver

#### INFECTIOUS DISEASES

Specific infections may involve the liver, some affecting the parenchyma, some the reticuloendothelial system, and some the connective tissue. The liver may become fatty or cirrhotic. The organisms causing the general infection may be found in the liver.

peripheral neuritis meningeal symptoms or residual renal damage Iritis iridocyclitis and uveitis may develop after weeks or months

### Diagnosis

Confronted with a case showing fever leukocytosis and meningeal symptoms the first thought would be of meningitis In mild cases an upper respiratory tract infection or food poisoning might be suspected The absence of jaundice will make the diagnosis even more difficult In a warm climate or in summer in a patient possibly working or playing in contaminated water and in a neighborhood where the disease is known to occur there should be a suspicion that the case is one of leptospiral disease Finding of the organisms in the blood in the first stage and in the urine in the second stage will determine the diagnosis Later the agglutination tests will be positive

### Treatment

The most important part of the treatment is supportive and dietetic management such as has been outlined under infectious hepatitis (p 492) Vaccines and serums have been shown to be of some help Various antibiotics have in addition been used Some clinicians have reported great amelioration or entire cure of an attack after the administration of penicillin in daily doses of one million units or more if given in the first stage and even some benefit when given later Others have claimed that Aureomycin in doses of 0.25 to 0.5 gm every six hours by mouth or 200 to 500 mg intravenously in severe cases is twice as good A later study by another group trying out these and three other newer antibiotics claims that no definite benefit was shown after administering any of these antibiotics

*Symptomatic treatment* is indicated such as oral hygiene analgesics for pain enemias for constipation extra vitamin K for bleeding and methenamine and sodium biphosphate for dysuria Serious renal damage may require heroic measures meningeal symptoms may require a spinal tap

We are fortunate that this disease is rare in our country

### TUBERCULOSIS

In pulmonary tuberculosis there is a tendency to develop a fatty liver In long standing cavitation with secondary infection or with suppurative tuberculous lesions anywhere amyloid liver will develop (see pp 516 and 617) Sarcoidosis is another complication Miliary tuberculosis will occasionally show multiple tubercles in the liver The findings of the primary disease with evidence of liver damage will suggest the type of liver involvement Treatment consists in the measures for tuberculosis and the diseased liver

body through defects in the skin abrasions in mucous membranes and even the conjunctivæ. The infection has been acquired by swimming in water contaminated by urine or feces of infected animals and by the drinking of such water. It is seen predominantly in men. The incubation period is four days to three weeks.

### Symptomatology

The symptoms can be divided into three stages as follows.

The *initial febrile stage* known as the bacteremic stage with temperatures ranging from 101 to 104° F. may last from two to nine days. The onset is abrupt often with a chill fever severe headache prostration and muscular pains. The gastrointestinal involvement will produce symptoms of a gastroenteritis pulmonary involvement cough expectoration and evidences of patchy consolidation. Examination will disclose conjunctivitis upper respiratory infection herpes signs of meningeal irritation the lung findings and tenderness of involved muscles. The abdomen is usually soft and the liver may not be palpable. There is evidence of beginning renal disease oliguria albuminuria and urea retention. The cerebrospinal fluid may show increased protein and cells. A leukocytosis of 20 000 or more is usual. The blood is teeming with the organisms visible on darkfield examination.

The *second stage* called the toxic or icteric stage although icterus does not always occur follows the subsidence of fever and lasts about a week or ten days. Ninety per cent or more of cases show icterus hepatomegaly and occasionally splenomegaly the remaining 10 per cent will have other symptoms. A well defined toxic stage occurs in 50 per cent of cases. There may be bleeding from mucosal surfaces or under the skin. Cerebral as well as meningeal symptoms may be present headaches restlessness stupor and even coma occurring at times. The leukocytosis may go to great heights the urine becomes more scanty and contains much bile (in icteric cases) albumin and hyaline and granular casts and may show sugar. Urea retention and high blood pressure may occur patients occasionally going into uremic coma. Symptoms of pancreatitis may complicate the picture. At this stage serum agglutination tests show the development of antibodies. During the week or ten days of this stage many of the patients will die. In young patients under forty years of age the mortality rate is between 15 and 20 per cent in those over forty it is much higher having been recorded in epidemics from 40 to 60 per cent. In nonicteric cases few deaths have occurred.

*The Third Stage* If the patient is going to recover he goes into the third or convalescent stage during which all symptoms and signs gradually subside. The organisms disappear from the urine the agglutination titer advances and the patient gradually recovers although he may be weak for months. Some patients have sequelæ such as endocarditis.

required During the attack the diet as recommended for liver damage is indicated (p 481)

### PSITTACOSIS

At times psittacosis shows liver enlargement from passive congestion The liver involvement is treated as in any liver disease

### PARASITIC DISEASES AFFECTING THE LIVER

In tropical regions parasites invade the liver fairly frequently but in the temperate zone they are rarely encountered Amebic hepatitis and abscess and hydatid cysts discussed previously belong in this group Other parasitic infestations that have been mentioned as possibly invading the liver include the protozoa roundworms tapeworms trematodes or flukes and rarely coccidia These are discussed in the general chapter on parasites In schistosomiasis and leishmaniasis particularly the liver should be watched carefully for the development of hepatitis fatty changes cirrhosis and hepatic granulomas and suitable treatment should be instituted in addition to the destruction or elimination of the parasites (see parasites p 134)

### SYSTEMIC MYCOSES AFFECTING THE LIVER

Although the fungi of the various mycoses may invade most of the tissues of the body none have a predilection for the liver The two that have been identified however rarely are actinomycosis and histoplasmosis (see chapter on Mycotic Diseases p 623)

#### Actinomycosis

This disease is rarely found in the liver The ray fungus has been thought to originate in the ileocecal region but it has been demonstrated recently that it is absorbed from gingival pockets It may be carried to the liver through the portal vein or there may be direct infection from nearby structures such as the right lung The abdominal wall is frequently involved In the liver small abscesses are usually formed which coalesce and form a large bright yellow abscess often secondarily infected with pyogenic organisms As in abscesses of other types the liver tissues are damaged and a perihepatitis may occur with sinuses communicating with the peritoneal or pleural cavities or the abdominal wall

**Diagnosis** The symptoms of the initial lesion combined with symptoms of hepatitis and the finding of an enlarged liver or of a liver tumor or abscess should occasion suspicion of actinomycosis Demonstration of the ray fungus in discharges from any of the lesions will determine the diagnosis

**Treatment** Actinomycosis was formerly considered incurable but

## SYPHILIS

Although syphilis is becoming a comparatively rare disease it is still to be regarded as the possible cause of liver disease (see p 621). In congenital syphilis the liver is swarming with spirochetes which may produce severe damage to the parenchyma and interstitial tissues. In early acquired syphilis there may be an acute hepatitis in a small percentage of cases which usually clears up rapidly under adequate anti-syphilitic treatment. Untreated cases may be followed by cirrhosis or may terminate with an acute yellow atrophy. In the late stage of syphilis gummas are not uncommonly found and may resemble neoplasms. These may go on to necrosis and parenchymal damage followed by scarring and cicatricial contraction with marked deformities of the liver (hepar lobatum).

## Diagnosis

The symptoms are those of hepatitis or cirrhosis. The irregular nodular liver may be palpated. Positive serologic findings and the presence of other syphilitic lesions help to clinch the diagnosis.

## Treatment

There should be suitable antisyphilitic treatment with avoidance of arsenical compounds which might further damage the liver. The hepatic involvement requires the same care that has been outlined for any form of liver damage. If not too much parenchymal damage has been done and there has been adequate regeneration of functioning liver tissue the patient will get reasonably well.

## TYPHOID FEVER

This disease occasionally involves the liver either in the form of focal lesions resembling miliary tubercles throughout the parenchyma or through the lymphatics in the portal area. Fatty liver is also liable to occur especially if a balanced diet with adequate vitamin and mineral supplements is not carried out. It usually clears up with recovery from typhoid infection.

## INFECTIOUS MONONUCLEOSIS

This disease is regularly accompanied by the lymphocytic infiltration of many organs including the liver. The liver involvement differs from that of infectious hepatitis in that there is minimal parenchymal damage and no architectural change connected with the lymphocytic infiltration. The symptoms are those of infectious mononucleosis in general: pharyngitis, adenopathy and fever accompanied by painless jaundice. Blood and serologic studies make the diagnosis. The liver condition clears up rapidly with the subsidence of the disease so that prolonged bed rest is not

ascitic fluid will be reabsorbed. In *pneumococcal pneumonia* cardiac failure is the cause of enlargement and of passive congestion.

In long standing *chronic decompensation* the "nutmeg liver" will develop and there may be resulting fatty or cirrhotic changes. With pulmonary infarction the icterus may be confusing and may be assumed to have come from the enlarged liver. It may result from either. Every effort must be made to establish compensation. In either case the treatment of the fatty or cirrhotic liver must not be neglected.

*Aneurysm of the hepatic artery* is a rare condition. The size varies from 1 to 5 or 6 cm. or more in diameter. Practically all these aneurysms are arteriosclerotic in origin although trauma has been mentioned as a cause. Pressure on surrounding tissues may induce symptoms varying from pain and retrostaltic gastric symptoms to jaundice from common duct pressure. Rupture with hemorrhage may occur into the biliary tract, stomach or duodenum causing hematemesis and melena. The diagnosis is confusing especially since it may be complicated by peptic ulcer or biliary tract disease. The finding of a pulsating tumor over which a hum can be heard is of great value. X-ray studies may show pressure effects or a tumor. Treatment consists in ligation of the artery.

### Trauma of the Liver

The liver may be injured in a number of ways producing various lesions. Trauma may result from

1. Crushing injuries to the abdomen as in train and automobile accidents especially steering wheel injuries or blows, kicks or objects falling on and deeply compressing the abdomen.
2. Penetrating wounds such as bullet or knife wounds from without or needles, fish bones or other sharp articles which were swallowed and have perforated through the gastric or intestinal wall.
3. Gallstones perforating through the gallbladder or ducts and lodged in the liver.
4. Diagnostic procedures such as liver biopsy either by the blind punch method or at operation or accidental perforation during a right pleural tap.
5. Surgical operation either on the liver itself for removal of tumors or of parts of the liver or accidentally during operations on neighboring organs.

### Pathology

With crushing injuries the liver may be only contused with its capsule intact producing a hyperemic edematous area which may result in chronic hepatitis. At times the right diaphragm may be torn permitting the liver to extend upward partly into the chest. When the liver tissue is torn there is usually bleeding. With the capsule intact this may pro-

favorable results have been obtained with large doses of antibiotics. Penicillin one million units or more every four hours, broad spectrum antibiotics in comparable doses, and massive doses of sulfonamides have caused remissions in many cases. They should be used to obviate the need for operation or used after operation if this has been required. Increasing doses of iodine such as potassium iodide may promote absorption of exudate. Mortality rates as low as 20 per cent have been recently reported.

### Histoplasmosis

Hepatomegaly, splenomegaly, emaciation, leukopenia and irregular fever are the prominent findings in histoplasmosis which is a rare infection. Since all these findings occur also in cirrhosis the specific diagnosis is rarely made before death. The causative organism *Histoplasma capsulatum* can be found in sternal marrow, in peripheral blood and in material obtained by splenic puncture. Smears from lymph nodes removed at biopsy and from ulcers of the skin and mucosa may also reveal the organism. Various treatments have been recommended with some good results obtained by thorough treatment with antibiotics and sulfonamides.

### COLLAGEN DISEASE

The various manifestations of collagen disease are discussed in the general chapter (p. 99). At autopsy the pathologist at times can demonstrate microscopically in the liver changes such as polyvasculitis, the presence of deposits resembling the collagen substance of the L.E. cell and some evidence of fibrinoid degeneration and necrosis, all indicative of so called collagen disease. Their presence however is not usually associated with symptoms of liver disease. Rarely the liver may be enlarged and liver function tests may show abnormalities, an elevated serum globulin, especially gamma globulin being most frequently observed. In the presence of evidences of one or more of the collagen diseases elsewhere, these liver abnormalities can be suspected of being a part of the general picture. The treatment is that of collagen disease in general.

### CARDIOVASCULAR DISEASES

*Cardiac decompensation* by causing congestion of the liver may produce both acute and chronic changes. In an *acute decompensation*, especially of the right side of the heart, the liver may become markedly congested and enlarged, its outline not firm but often tender. There may be symptoms of portal hypertension, even ascites, and congestion in the gastrointestinal tract will cause symptoms. When compensation is re-established the liver will rapidly shrink to its normal size, all symptoms of hepatic and gastrointestinal congestion will disappear rapidly and

ascitic fluid will be reabsorbed. In *pneumococcal pneumonia* cardiac failure is the cause of enlargement and of passive congestion.

In long standing *chronic decompensation* the "nutmeg liver" will develop and there may be resulting fatty or cirrhotic changes. With pulmonary infarction the icterus may be confusing and may be assumed to have come from the enlarged liver. It may result from either. Every effort must be made to establish compensation. In either case the treatment of the fatty or cirrhotic liver must not be neglected.

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duce a hematoma which may be absorbed may produce a cyst or become infected resulting in abscess. When the capsule is torn either from the crushing injury or from foreign bodies blood and often bile from the liver injury may pour directly into the peritoneal cavity or into the retroperitoneal space and result in sudden death or if only small in amount may cause some peritonitis. Secondary infection with liver abscess and peritonitis is a serious complication. Trauma may cause injury to an aneurysm of the hepatic artery which may rupture and finally erode into the biliary tract blood passing into the duodenum and causing melena or even hematemesis.

Any trauma resulting in occlusion of the portal vein causes rapid accumulation of ascitic fluid hemorrhage with tarry stools ileus and severe abdominal pains rapid enlargement of the spleen and death within a few days. Extensive thrombosis of the portal vein also results in death but not so rapidly.

### Diagnosis

With a crushing abdominal injury or a puncture wound the liver should be thought of in considering whether or not any organs have been injured. A slight injury to the liver may produce only mild symptoms some pain and retrostaltic symptoms which may subside with rest. In a case with a hematoma formed under the capsule or by a walling off against another organ absorption of blood will produce hemolytic jaundice later confusing the picture with a hepatitis. A severe injury will produce shock evidences of blood loss severe pain usually over the liver tenderness and rigidity in the right upper quadrant a fluid wave and all the symptoms of an acute abdomen. Differentiation from injuries to other organs or their coexistence will be almost impossible except on surgical exploration although x-ray films showing absence of free air in the peritoneal cavity will contraindicate perforation of a hollow viscus in most cases.

### Treatment

Except in the mild case exploratory operation is indicated. Lacerations may be sutured bleeding stopped by a ligature or a Gelfoam pack and fragments or even badly damaged parts of the liver may be removed. Medical care as for any liver disease is important (see Liver p. 480).

### Prognosis

With prompt operation the mortality rate should not be too high except in cases with large severe injuries.

### Nontraumatic Rupture of the Liver

Spontaneous rupture of the liver is a rare condition. The few dozen

cases which have been reported have shown it to be a complication of a variety of diseases either directly or indirectly affecting the liver. The probable immediate cause is an extreme ischemic infarction due to occlusion of an artery in the liver with or without occlusion of the portal vein. At times undue exercise or an epileptic convulsion has preceded the rupture. As in the case of injury the rupture may take place with or without rupture of Glisson's capsule. As a rule there is severe pain at the time of rupture. In the cases in which Glisson's capsule has not ruptured and a hematoma has formed within the liver or in which a hematoma has been formed as a result of spontaneous walling off or of hemorrhage only into the lesser sac the subsequent symptoms may be mild often with jaundice due to breaking down of the blood. Most cases with rupture of the capsule and massive bleeding into the peritoneal cavity will have the same symptoms and require the same treatment as that for traumatic rupture described in the previous paragraphs.

## The Gallbladder and Bile Ducts

### General Discussion

#### ANATOMY

This part of the biliary tract is concerned with the collection and temporary storage of the bile and the delivery of bile and pancreatic juice to the intestine. Figure 94 shows the anatomy and the relationships of the various parts of this system. The most important anatomical factors are as follows:

- 1 The gallbladder and ducts lie mostly beneath the liver.
- 2 The gallbladder points upward, backward and to the left.
- 3 The fundus or wide end of the gallbladder, its lowest part, extends usually to or a little below the margin of the liver, often touching the abdominal wall; the second portion or body comes in contact with the duodenum and proximal transverse colon.
- 4 The neck, S-shaped, tapers into the cystic duct, which contains spiral valves.
- 5 The extrahepatic bile ducts lie in close relationship with the portal vein, hepatic artery and hepatic vein.
- 6 The common duct lies in contact with or passes through the head of the pancreas.

duce a hematoma which may be absorbed may produce a cyst or become infected resulting in abscess. When the capsule is torn either from the crushing injury or from foreign bodies blood and often bile from the liver injury may pour directly into the peritoneal cavity or into the retroperitoneal space and result in sudden death or if only small in amount may cause some peritonitis. Secondary infection with liver abscess and peritonitis is a serious complication. Trauma may cause injury to an aneurysm of the hepatic artery which may rupture and finally erode into the biliary tract blood passing into the duodenum and causing melena or even hematemesis.

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### Treatment

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### Nontraumatic Rupture of the Liver

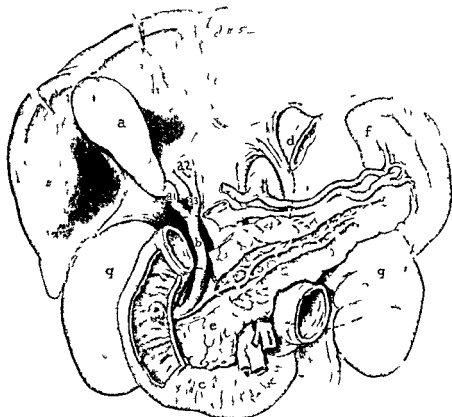
Spontaneous rupture of the liver is a rare condition. The few dozen

There is a delicate balance of pressure in the biliary tract. Pressure is greatest in the hepatic duct representing the secretory pressure of the liver. When the sphincter of Oddi is closed as it is when the duodenum is empty, pressure in the common duct increases and the bile unable to overcome the pressure in the hepatic duct is forced into the cystic duct and gallbladder to be stored until required for the digestion of fat in the duodenum. A delicate mechanism at first thought by Meltzer to be due entirely to contrary innervation but later demonstrated by Ivy to be due to the hormone cholecystokinin governs the filling and emptying of the gallbladder. Cholecystokinin is elaborated in and absorbed from the duodenum in the presence of fats and to some extent fruit juices and peptones. It induces the contraction and emptying of the gallbladder coincident with the relaxation of the sphincter. The pressure in the gallbladder during this contraction is normally greater than the pressure in the common duct but never appreciably greater than that in the hepatic duct so that bile flows steadily to the duodenum. However a little gallbladder bile may regurgitate into the hepatic ducts as shown in cholecystograms.

Bile salts absorbed from the duodenum stimulate the secretion of bile and increase the secretory pressure in the hepatic ducts. Theoretically the result of entire abstinence from eating for twenty-four hours would result in great overdistention of the gallbladder by the liter or more of bile it would accumulate. To prevent this distention the gallbladder concentrates the bile it is storing up to eight or ten times its original concentration. This provides a more potent bile for the digestion of the fats in the duodenum which have stimulated its expulsion from the gallbladder.

#### PATHOGENESIS

Although the motor function of the biliary tract is regulated by the mechanism just described it may be affected to a less degree by extraneous factors such as changes in intra-abdominal pressure in pregnancy, ascites or large abdominal tumors or as a result of coughing, laughing or sneezing. Infections, psychic influences, endocrine disturbances and anaphylactic shock also upset the normal mechanism. Only a few drugs have a direct effect on the motor function. The effect of drugs stimulating the sympathetic and parasympathetic systems is uncertain and variable and therefore confusing. Histamine may increase the tonus of the gallbladder and sphincter. Morphine and to a less extent other opium derivatives rapidly and markedly increase the pressure in the biliary tract which it is contended is due partly to increase in tonus and contraction of the sphincter of Oddi. The nitrites antagonize this spastic effect. Magnesium sulfate in contact with the duodenal mucosa will produce the same effect as fats i.e. relaxation of the sphincter and con-



*Figure 94* Anatomy of gallbladder and ducts. Liver retracted. The stomach is indicated by dotted line. *a* Gallbladder *a1* cystic duct *a2* two hepatic ducts *a3* common hepatic duct *b* common duct *c* duodenum *d* esophageal opening of stomach *e* pancreas *f* spleen *g* kidneys (Courtesy of Wyeth Laboratories)

### PHYSIOLOGY

The mechanism for the disposal of the bile is not intricate. Bile is secreted by the liver continually day and night the total amount being variously estimated as from 800 to 1500 cc in twenty-four hours. The bile contains (1) excretory material cholesterol, fats, calcium and the bile pigments formed by the breaking down of blood cells in the liver and (2) secretory substances the bile acids, bile salts and alkalis which aid in the digestion and absorption of fats in the diet. The two ducts from the right and left lobes of the liver merge to form the hepatic duct which in turn merges with the cystic duct to form the common duct. This is usually joined by the pancreatic duct just before entering the duodenum although each may enter it separately. The entrance of bile into the duodenum is guarded by a sphincter-like arrangement of the circular muscles of the duct collectively called the sphincter of Oddi, the opening and closing of which is regulated by the presence or absence of food especially fats, in the duodenum.

center of a stone was presented as proof of this hypothesis. Now, however, such bacteria are rarely if ever found in stones, and except in acute secondary infections, gallbladder bile usually proves to be sterile with no evidence demonstrated of infection of the gallbladder wall. There is no doubt, however, that chronic or repeated infections or allergic reactions in the gallbladder and ducts or marked duodenal irritation and inflammation may cause sufficient organic changes to interfere with proper function to produce stasis and to favor the formation of stones.

The well known frequent incidence of gallbladder disease and gallstones in pregnancy has been attributed to the hypercholesterolemia occurring in the later months, to change in intra-abdominal pressure, corsets and "physiologic dyskinesia." However, careful histories of such cases will disclose severe nausea, vomiting and anorexia with particular dislike for fatty foods and decreased frequency and quantity of food ingested to account for excessive stasis and concentration of bile in the gallbladder. A history of the same dietary deficiencies will be found in obese patients starving to reduce weight, in psychiatric patients refusing food and in patients with endocrine disturbances such as diabetes and hypothyroidism or with chronic diseases of various kinds when diets have been neglected. I believe that it is safe to say that with the exception of cases in which mechanical factors as mentioned above interfere with drainage from the gallbladder, the formation of gallstones is almost invariably due to insufficient frequency of intake of fats producing stasis of bile in the gallbladder with concentration to the point of precipitation of or caking out of its constituents. The time required for the formation of even large stones may be measured in weeks or months.

The normal concentration of cholesterol in the gallbladder is so much greater than that ever attained in the blood that it seems ridiculous to consider that hypercholesterolemia may be a factor in the formation of gallstones. Furthermore, the fat-free diet often recommended in cases of biliary tract disease, as well as in arteriosclerosis, has been shown not to have any appreciable effect on blood cholesterol, on the progress of arteriosclerosis or on the formation of gallstones. Blood cholesterol concentration is regulated independently of fat ingestion. (See discussion on cholesterol in chapter on Liver, p. 463.)

#### PATHOLOGY

The gallbladder and ducts, all a part of one small system, are usually affected together, rarely separately. Acute and chronic cholecystitis is usually accompanied by more or less duct inflammation and frequently by gallstone formation. The liver and its capsule and the pancreas are almost invariably affected to a greater or lesser degree. Neoplasms may occur in any part of the tract, the malignant ones being either primary or secondary. Fistulas may be found between the gallbladder and ducts.

traction of the gallbladder. The many other drugs recommended and tried in diseases of the biliary tract have been shown to have no direct or consistent effect. Cholagogues, however, such as bile salts, which increase the flow of bile to some extent, thereby increase pressure in the hepatic ducts and probably to some extent in the entire biliary tract.

In the case of narrowing, malformations or diseases of the biliary tract, more or less marked changes in function are of course to be expected. It is well to remember in disease that fats, especially emulsified fats such as cream and egg yolk, promote relaxation of sphincter spasm and thus encourage biliary drainage better than any drug except perhaps magnesium sulfate. They have the advantage of ease and frequency of administration when combined with other nutrients necessary for adequate nutrition.

### Formation of Stones

The mechanism of concentration of bile is the important factor in the formation of gallstones. Normally the concentration increases constantly up to eight to ten times while the bile remains in the gallbladder, largely owing to absorption of water, sodium chloride and bicarbonate. This causes an increasing concentration of bile salts, pigments and cholesterol and a progressive decrease in the pH. The hitherto pale, yellowish bile becomes dark and viscid. The bile salts, mainly taurocholates and glycocholates, are absorbed only from a diseased gallbladder; normally they enter the duodenum.

In prolonged stasis, concentration of bile pigments may reach fifteen times or more the strength of liver bile. Occasionally, especially when combined with calcium, these concentrated pigments separate out and form irregular pigment stones. These are often called "mulberry stones" in the gallbladder or the "earthy stones" in the bile ducts. The concentration of cholesterol, aided by alteration of the cholesterol/bile salt ratio, may increase to the point at which it results in the formation of the pale, usually single, smooth or slightly granular *cholesterol stone*, or it may infiltrate the gallbladder mucosa, producing cholesterosis or *strawberry gallbladder*. Calcium, usually present in the form of the carbonate, may be precipitated out of the bile in the form of *milk of calcium bile*. It also may produce small, hard *calcium stones*, may act as a coating on cholesterol stones, or may infiltrate the bladder wall, producing the calcified gallbladder. Calcium is usually combined with cholesterol and pigment to form the common multiple faceted *mixed stones*, constituting 80 per cent of all calculi.

It seems entirely reasonable to suppose that gallstones are formed simply from excessive concentration of bile due to its prolonged stay in the gallbladder. Formerly it was considered that infection played a part and the occasional finding of a typhoid bacillus or other organism at the

stones at operation when the patient has had no pain for so long a time as to have forgotten it

### Jaundice

In the case of stones unable to pass through the common duct into the duodenum or impacted in the walls of the duct or obstruction from other causes jaundice is a common finding either constant or intermittent depending upon the degree of persistence of the obstruction. Accompanying pruritus is often but not always indicative of obstruction. It may precede the development of icterus. It is important to note the relation of the onset of symptoms to the occurrence of acute generalized infections, focal infections and intestinal infestations. It is equally important in a patient with a history of biliary tract disease to be on the lookout for complications such as perforations, obstructions and metastases. The specific symptoms of the separate diseases will be discussed together with their characteristic pathologic findings and their treatment.

## EXAMINATIONS

### Physical Examination

Examination may show nothing abnormal or may elicit tenderness, rigidity or a mass in the right upper quadrant and enlarged liver and spleen or evidences of an acute abdomen. A Riedel lobe may be mistaken for the gallbladder. In all cases a careful general physical examination is essential to rule out organic disease elsewhere as in the heart, lungs, pelvis and spine which may cause similar symptoms. Cholecystitis may produce electrocardiographic changes. A careful search for any possible focal infections such as devitalized teeth, infected tonsils and sinuses, pelvic and urinary infections should also be carried out.

### Peritoneoscopy

This has been performed in patients in whom the diagnosis is uncertain. However, the limited view of the biliary tract which can at best be obtained by this method makes it inadvisable to do this type of examination.

### Laboratory Examinations

In all cases some laboratory examinations will be necessary in order to make a diagnosis. The following procedures will prove to be of great value.

1. *Blood studies* should include complete blood cell counts, alkaline phosphatase, sedimentation rate, chemical and serologic determinations and prothrombin, coagulation and bleeding time.

2. *Liver and renal function tests* as indicated.

3. *Fractional gastric analysis* after histamine injection not only to determine acidity which may be altered but also to estimate emptying



or between either the gallbladder or ducts and neighboring organs or the skin. The pathology of each type of lesion will be discussed in more detail together with the diagnosis and treatment of each.

#### SYMPTOMATOLOGY

In biliary tract diseases in general even in the presence of stones, there may be no symptoms at all or they may be so mild as to be disregarded by the patient. *Referred symptoms* are common. The presence of the adjacent disease may irritate the pyloroduodenal region or the colon. In the former case the usual *retrostaltic* or *reflex symptoms* will be produced including epigastric discomfort, anorexia, belching, heart burn, eructations, nausea, regurgitation or even vomiting. All these symptoms usually occur soon after eating. There is usually an aversion to fats probably due to the feeling that they cause symptoms. Irritation of the adjacent colon may cause increased motility as evidenced by a tendency to *diarrhea* or spasms with gaseous distention as evidenced by cramps and expulsion of *flatus*.

Local symptoms are usually mechanical in origin. They may be mild with only a feeling of a "lump" or stone in the right upper quadrant. *Pain* may vary in severity. It is usually located in the gallbladder region with characteristic radiation around the right side to the right subscapular region. The severity and direction of radiation vary in different patients. The pain may radiate directly upward to the precordium or may go to the left side or to the lower abdomen. Also occasionally the pain and radiation may be entirely on the left side even though the gallbladder is not transposed. Mild or moderate pains may be due simply to the presence of diseased or distended gallbladder or ducts or may be caused by gallbladder contractions. When the pains occur intermittently with chills and fever (Charcot's fever) it suggests an added acute infection with intermittent obstruction.

#### Biliary Colic

Typical *biliary colic* the pains of which have been described as being as severe as labor pains has been shown to be due to the attempted passage of a stone through the neck of the gallbladder. Careful serial x-ray studies by Lewis Gregory Cole showed one day a gallbladder and cystic duct each containing a few easily counted stones. At this time the patient had no symptoms. The night following this study the patient had an attack of severe typical colic and returned the following morning with no more pain. X-ray films then disclosed one stone missing from the gallbladder and one added to the cystic duct. This observation showed that stones in the gallbladder and cystic duct may cause no pain the pain being due only to the passage of the stone through the neck of the gallbladder. It explains the frequent finding of cystic and common duct

stones at operation when the patient has had no pain for so long a time as to have forgotten it

### Jaundice

In the case of stones unable to pass through the common duct into the duodenum or impacted in the walls of the duct or obstruction from other causes jaundice is a common finding, either constant or intermittent depending upon the degree of persistence of the obstruction. Accompanying pruritus is often but not always indicative of obstruction. It may precede the development of icterus. It is important to note the relation of the onset of symptoms to the occurrence of acute generalized infections, focal infections and intestinal infestations. It is equally important in a patient with a history of biliary tract disease to be on the lookout for complications such as perforations, obstructions and metastases. The specific symptoms of the separate diseases will be discussed together with their characteristic pathologic findings and their treatment.

## EXAMINATIONS

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2. Liver and renal function tests as indicated.
3. Fractional gastric analysis after histamine injection not only to determine acidity which may be altered but also to estimate emptying

time and to look for admixtures such as blood with regurgitated duodenal contents

4 *Examination of duodenal contents* which can immediately follow the gastric analysis or can be done separately, should include simple aspiration followed by aspiration after the injection of 75 cc of 33 per cent magnesium sulfate solution. The finding of cholesterol or calcium bilirubinate crystals may suggest the presence of gallstones. Recovery of blood and especially of cells examined by the Papanicolaou method may indicate biliary tract malignancy. Occasionally ova or parasites such as *Lambia intestinalis* may be found. Cultures are of little value, being subject to much contamination.

5 *Stools* examined for blood, bilirubin, urobilin, pancreatic ferments and parasites or ova may give valuable information.

6 *Urine examination* is important. The finding of bilirubin may show early biliary obstruction. If urobilinogen is found, it rules out obstruction.

7 In the presence of *jaundice* the following tests will be of value for differentiation between extrahepatic biliary obstruction and hepatitis: (1) The alkaline phosphatase will rise steadily in obstructive jaundice and may be high early in hepatocellular jaundice and then subside. (2) The serum bilirubin will rise rapidly in the hepatocellular form, remain steady and later go down, but will rise steadily in obstructive jaundice. (3) Other liver function tests such as cephalin flocculation and thymol turbidity and flocculation, blood protein partition and prothrombin tests will point to liver damage. Low or absent urobilinogen in the urine to obstructive jaundice.

### X-ray Examination

These studies should include not only cholecystography or cholangiography, but also a complete gastrointestinal series and a barium enema to rule out other diseases that cause symptoms or complicate the gallbladder disease. In the early films after a barium meal the ampulla of Vater may be seen filled with barium and an enlarged papilla may cause a defect on the inner side of the second portion of the duodenum. Opaque stones may be seen in relation to the cap (Fig 95 a). Chest, urological and other x-rays may also be indicated, especially if operation is contemplated. One or more plain scout films preceding the other examinations may show opacities due to stones or air in the ducts or gallbladder when perforation has occurred. Gas collections may give a hint as to some diseases. Formerly much importance was attached to the gallbladder which could be visualized in scout films. It has been shown that similar shadows can be ducts, a filled duodenal cap or a Riedel's lobe. Studying the gallbladder region in films of the stomach or intestine after a barium meal or enema may show stones or a calcified gallbladder (Fig 95 b).

*Oral Cholecystography* The techniques of preparation and carrying

out of this study are being changed so frequently that detailed instructions must be left to the person doing the x rays. In general the principles involved consist in giving to the patient in the evening tablets of the agent to be used for opacification of the gallbladder and ducts followed by abstinence from all foods and even liquids until films of the gallbladder region are obtained the following morning. If the so called dye has been absorbed if the liver has excreted it in the bile if the sphincter of Oddi is functioning and the ducts are patent the bile with its opaque iodine contents will be carried into the gallbladder which will then cast a shadow on the films. These films are taken to determine (1) the size shape and location of the gallbladder (Fig 96 *a*) (2) the density and homogeneity of the gallbladder and ducts (3) areas of increased density due to stones or accumulations of inspissated bile ("muck") containing calcium (Fig 96 *b*) (4) areas of decreased density in the gallbladder and ducts (Fig 96 *c*). These may be due to nonopaque stones or neoplasms. Both opaque and nonopaque stones will be shown in different locations when the position of the gallbladder is changed by taking films in various positions standing and lying down. One or more smooth or irregular areas of decreased density which remain fixed at one location in spite of changes in position usually indicate neoplasms most frequently papillomas attached to the gallbladder wall. Small adenomas may be missed since they are usually accompanied by gallstones. Cancer when large produces nonvisualization when small it is obscured by stones.



Figure 95 *a* Gallstones in relation to duodenal cap. Barium meal study, lateral film. 1 Opaque stones in gallbladder. 2 duodenal cap. 3 antrum. *b* Calcified gallbladder containing stones. 2 duodenal cap. 3 descending duodenum.



Figure 96 a Gallbladder located high under the ribs, transversely placed. Cholecystogram Homogeneous, no stones b Gallstones, standing position. Cholecystogram gallbladder large and long extending into the pelvis. Multiple nonopaque stones clearly visible c Cholecystogram 1 Opaque stones in gallbladder 2 stone in common duct 3 dilated common duct

**POSTPRANDIAL FILMS** These are made after the filled gallbladder has been studied. The patient is given a small meal high in fat content or some commercial preparation such as Cholex which contains fat, gelatin and lecithin. This provides a physiologic stimulus to gallbladder emptying. Films taken twenty and forty minutes afterwards and later if desired normally show some increase in density (concentration) and decrease in size (emptying) of the gallbladder and it may empty entirely. Failure to concentrate or expel the bile may be due to functional or organic disturbances of the gallbladder or ducts. At times stones not seen when the gallbladder is filled will become visible when it gets smaller. Usually when the gallbladder empties and sometimes even before this the ducts filled with opaque bile will become visible. Today we expect to see clearly the cystic duct, the common duct and often also the hepatic ducts (Fig 97). Abnormalities of the ducts such as stones, neoplasms, narrowing or obstruction may become visible.



Figure 97 a Normal gallbladder and ducts postvacuumation film 1 Dense gall bladder shadow 2 neck of gallbladder 3 cystic duct spiral valves visible 4 common duct b Gallbladder with constriction postprandial film 1 Gallbladder showing constriction 2 neck of gallbladder 3 cystic duct spiral valves clearly seen 4 common duct 5 hepatic duct



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routine careful exploration. Such x rays can of course also be done later if a "T" tube drain has been left in. Many surgeons now regard cholangiography as part of a gallbladder operation but some object to the time and risk involved.

*Cholecystography and cholangiography* will at times give valuable information about the nature of calcific areas found in scout films (Fig 100). Such areas may be shown to be within the gallbladder or outside of it in the kidney mesentery or the lumen of the gastrointestinal tract. Barium by mouth or by enema can be used in conjunction with cholecystography and cholangiography not only to pinpoint the opacities but also to indicate whether pressure deformities of the stomach, duodenum or transverse colon are due to pressure or adhesions to the gallbladder or ducts.

**WARNING** Rarely patients will be found who are allergic to the agent used for visualization especially to its main constituent iodine. Violent allergic reactions may follow not only in the skin but also in the



Figure 98. *a* Normal cholangiogram (intravenous). 1 Cholangiogram in hepatic radicles. 2 Cholangiogram in duodenum. 3 Cholangiogram in common duct. *b* Intravenous cholangiogram. Dilated ducts due to stones. 1 and 2 Right and left hepatic duct. 3 Common duct. Arrows point to 5 calculi in distal part of common duct. Patient treated for 5 years for heart trouble. (Used through the courtesy of Dr. Lewis L. Immerman.)



**NONVISUALIZATION** If the gallbladder fails to be visualized after ingestion of the dye it indicates either that the dye has not been absorbed in the intestine or excreted by the liver or that the lumen of the ducts or gallbladder is obstructed. It is therefore important to remember that cholecystography should not be performed in the presence of jaundice when the liver is badly damaged or when intestinal absorption is faulty as in diarrhea. If none of these contraindications are present even failure to visualize the gallbladder should not be accepted categorically as evidence of disease or stones. In some cases intravenous cholecystography will serve to visualize the gallbladder when the oral method fails. In many cases a gallbladder diet rich in fats such as described hereafter (p 552) will clear the tract of inspissated bile and even of minute stones (less than 5 mm in diameter) and permit perfect visualization after a month or two. Such a gallbladder may then prove to be normal or may show stones or other abnormal findings.

**Intravenous Cholecystography** Originally all gallbladder studies were made after intravenous injection of various iodine containing chemicals excreted exclusively by the liver. Impurities in the chemicals frequently caused toxic symptoms. They were therefore abandoned when the oral method was shown to be satisfactory. In recent years new dyes have been prepared which have shown no untoward effects when given intravenously. By using them the whole gallbladder study can be carried out in a few hours in the morning. The interpretation of the findings is the same as with the oral method.

**Cholangiography** As has already been mentioned the bile ducts can be satisfactorily visualized by oral or intravenous cholecystography. When the gallbladder had been removed it was formerly considered impossible to visualize the ducts until it was discovered that with films taken during the first few minutes after intravenous injection of Cholografin the ducts could be beautifully seen and abnormalities noted (Fig 98). After this other investigators demonstrated that equally good results could be obtained when films were made during the first few hours after oral administration of one of the conventional substances. This has been a great help in the study of patients with symptoms after cholecystectomy. Serial tomograms are an aid to clarification. Cholangiography may also be of value in visualizing the bile ducts of patients who fail to show a gallbladder on cholecystography but it should not of course be done in a badly jaundiced patient.

**Postoperative Cholangiography** Immediately after operation upon the gallbladder while the abdomen is still open x ray films of the ducts can be made by injection of an opaque medium such as Lipiodol into a "T" tube inserted for drainage or by direct injection into the common duct through a hypodermic needle (Fig 99). This has proved to be a great help in finding stones in the ducts which were overlooked even with

routine careful exploration. Such x rays can of course also be done later if a "T" tube drain has been left in. Many surgeons now regard cholangiography as part of a gallbladder operation but some object to the time and risk involved.

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Fig 98 a Normal cholangiogram (intravenous). 1 Cholangiogram in liver radicles 2 cholangiogram in common duct 3 cholangiogram in duodenum b Intravenous cholangiogram dilated ducts due to stones. 1 and 2 Right and left hepatic duct 3 common duct 4 and 5 contrast in distal part of common duct. Patient treated for 5 years for heart trouble. (Used through the courtesy of Dr. Lewis L. Immerman.)



Figure 99 Cholangiogram at operation 1 Dilated common duct 2 site of stone 3 tube in duct

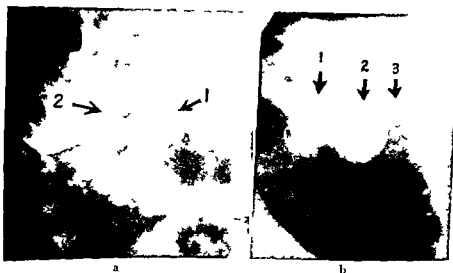


Figure 100 a Filled gallbladder relation to kidney stone 1 Gallbladder 2 kidney stone 3 calcified glands b Gallbladder with stone relation to calcified glands 1 Gallbladder and stone 2 and 3 calcified glands

nose and throat bronchi and gastrointestinal tract and even anaphylactic shock has been known to occur. It is therefore well in patients with allergies to inquire or test for iodine sensitization before cholecystography is started and to have hypodermic needles and epinephrine handy.

### DIFFERENTIAL DIAGNOSIS

Diseases of the biliary tract are many times mistaken for or complicated by a variety of other diseases. This makes careful study very important. The *retrostaltic* symptoms which may occur with biliary tract disease are also commonly found in diseases of the stomach and duodenum notably ulcer and cancer and in diseases of the intestines liver pancreas urinary tract and pelvis and even the heart.

The pains both mild and severe may be confused with those of a variety of other diseases. Not only are pains due to generalized *arterio sclerosis* aneurysm angina pectoris and coronary disease confusing but also these conditions frequently are found in patients with gallbladder disease. An attack of biliary colic may induce an attack of angina or actual infarction. Biliary tract pains should not be confused with *peptic ulcer pains* although both may occur together. Rarely a duodenal diverticulum may give similar pains. Colics due to small or large *intestinal obstruction* partial or complete may be due to coincident intestinal lesions or adhesions or may be the result of impaction of a gallstone in the small intestine usually at the ileocecal valve. Usually a history suggesting previous perforation of a gallstone may be elicited. Thickening of the ileocecal valve may cause similar symptoms. The pains of *renal colic* although characteristic as a rule may be atypical and confused with biliary colic. Pains due to *neurological* lesions such as the gastric crises of *cerebrospinal syphilis* the intense pains of herpes zoster and nerve pressure from spinal abnormalities or metastases are also at times a source of error. Ventral hernia either spontaneous epigastric or incisional hernia after operation also may cause pains which may be confusing as may esophageal hiatus hernia. Allergic reactions in the biliary or gastrointestinal tract or else here in the right upper quadrant must be watched for although the history of allergy and the rapid subsidence of symptoms especially after epinephrine ephedrine or antihistamines may be a help in the differentiation. Narcotic addiction must be ruled out. A crisis in sickle cell anemia may be confusing in a negro.

Jaundice is apt to be a deceptive symptom. After typical biliary colic if there have been previous similar attacks jaundice pretty well clinches the diagnosis of gallstones but does not rule out coincident cancer. Pancreatic lithiasis may give similar symptoms. Cancer of the head of the pancreas usually causes pain before onset of jaundice. *Painless jaundice* due to hepatic disease or to external pressure on the common duct or to diseases accompanied by hemolysis must always be borne in mind when



Figure 99 Cholangiogram at operation 1 Dilated common duct 2 site of stone 3 tube in duct



Figure 100 a Filled gallbladder relation to kidney stone 1 Gallbladder 2 kidney stone 3 calcified glands b Gallbladder with stone relation to calcified glands 1 Gallbladder and stone 2 and 3 calcified glands

grossly neglected and have become an important factor in the treatment of so-called intractable diseases. I insist upon clearing up all focal infections not only because of their etiologic significance but also to improve the general condition of the patient and to prevent complications especially if operation is being considered.

### Specific Treatment

In general the treatment of diseases of the gallbladder and ducts is based on two principles: first to encourage drainage and second to treat the disease.

**Drainage.** Drainage is mentioned first because it is frequently overlooked. If there is complete organic obstruction of the common duct drainage may be difficult or impossible. Even in such cases, however, application of physiological principles to promote drainage may be of value. We know that with relaxation of the sphincter of Oddi bile will flow into the duodenum and common duct pressure will be reduced. The presence of fat or oil or at times magnesium sulfate in the duodenum will cause even a badly spastic sphincter to relax. These substances can be introduced into the duodenum by feeding or by injection through a duodenal tube. If there is inflammation of the ducts and gallbladder there certainly can be no disadvantage in promoting such drainage except in the cases of suspected acute abdominal "calamity" in which operation is being considered. If the cystic duct or neck of the gallbladder appears obstructed repeated drainage may result in decrease in biliary pressure and encourage passage of small calculi (under 5 mm. in diameter) or inspissated bile. In cholecystitis with or without calculi drainage is always beneficial and tends to improve the tone of the gallbladder musculature so that a nonfunctioning gallbladder will often be restored to normal function. In disease of the intrahepatic or extrahepatic bile ducts the value of such drainage is obvious. The medical methods of promoting the drainage are as follows:

**NONSURGICAL BILIARY DRAINAGE.** This term was applied by Vincent Lyon to drainage of the biliary tract by continuous suction through a duodenal tube after the instillation of magnesium sulfate solution with peptone or olive oil added at times enabling him to remove large quantities of bile. Adequate drainage by this method can be obtained only by prolonged intubation or by frequently repeated passage of the duodenal tube. Either procedure is distressing to the sick patient and a burden on the attendants besides removing large quantities of bile which is required for normal gastrointestinal and biliary tract function.

**DRAINAGE BY DIET.** This method is more physiological and equally effective. It has the advantage of being capable of producing frequent or almost continuous biliary drainage without depleting the patient. Frequent feedings six or eight in twenty-four hours should consist of

the history is not typical. Their differentiation is discussed in the chapter on the Liver (p. 465).

*Pruritus* is usually an accompaniment of obstructive jaundice but its presence or severity is not indicative of the cause or the location of the biliary tract obstruction. It also occurs at times in hepatitis but not in hemolytic jaundice.

*Diarrhea* although a symptom of gallbladder disease may also be a symptom of many accompanying gastrointestinal diseases. All diarrheal cases require careful study to determine the origin of the symptom. Steatorrhea often accompanies obstructive jaundice.

An *enlarged liver* usually attributed to hepatic disease is frequently an accompaniment of disease of the gallbladder and ducts. There is always more or less liver damage in any case. An enlarged spleen may also be palpated at times.

The *gallbladder* may be palpated if inflamed occasionally during an acute episode and practically always with obstruction of the lower common duct. It is especially large in carcinoma of the head of the pancreas.

So far as the biliary tract is concerned a complete and careful x-ray study will usually clear up the diagnosis although failure of filling of the gallbladder may be a troublesome finding as mentioned under cholecystography. It is evident that only after a careful history and complete study of all suspicious regions can a definite diagnosis be made and the patient assured of adequate care.

## TREATMENT

### Prophylactic Treatment

*Prophylactic* treatment is most important and most frequently neglected. It should consist in promotion of normal biliary tract function and generally in the maintenance of a healthy mind in a healthy body. A normal diet consisting of three well balanced meals a day preferably with two or three additional feedings between meals and at bedtime has been shown to be essential for normal physiologic health including normal function of the gastrointestinal and biliary tracts. The caloric value of such a diet can be altered according to need but a normal balance of ingredients and frequency of feeding should be maintained. Fresh air, sunshine and provision for rest and recreation are of course important. The discovery by periodic health examination of any congenital or acquired abnormalities or diseases and their eradication are too much neglected. Especially focal infections should be prevented or cleared up if present. The role of devitalized or pyorrheic teeth, infected tonsils, chronic sinus infection, endocervicitis, proctitis, cryptitis and other pelvic and rectal infections in the production of chronic diseases has in recent years been de-emphasized. The result has been that such infections are being

grossly neglected and have become an important factor in the treatment of so-called intractable diseases. I insist upon clearing up all focal infections not only because of their etiologic significance but also to improve the general condition of the patient and to prevent complications especially if operation is being considered.

### Specific Treatment

In general the treatment of diseases of the gallbladder and ducts is based on two principles: first to encourage drainage and second to treat the disease.

**Drainage.** Drainage is mentioned first because it is frequently overlooked. If there is complete organic obstruction of the common duct drainage may be difficult or impossible. Even in such cases, however, application of physiological principles to promote drainage may be of value. We know that with relaxation of the sphincter of Oddi bile will flow into the duodenum and common duct pressure will be reduced. The presence of fat or oil or at times magnesium sulfate in the duodenum will cause even a badly spastic sphincter to relax. These substances can be introduced into the duodenum by feeding or by injection through a duodenal tube. If there is inflammation of the ducts and gallbladder there certainly can be no disadvantage in promoting such drainage except in the cases of suspected acute abdominal "calamity" in which operation is being considered. If the cystic duct or neck of the gallbladder appears obstructed, repeated drainage may result in decrease in back pressure and encourage passage of small calculi (under 5 mm. in diameter) or inspissated bile. In cholecystitis with or without calculi drainage is always beneficial and tends to improve the tone of the gallbladder musculature so that a nonfunctioning gallbladder will often be restored to normal function. In disease of the intrahepatic or extrahepatic bile ducts the value of such drainage is obvious. The medical methods of promoting the drainage are as follows:

**NONSURGICAL BILIARY DRAINAGE.** This term was applied by Vincent Lyon to drainage of the biliary tract by continuous suction through a duodenal tube after the instillation of magnesium sulfate solution with peptone or olive oil added at times, enabling him to remove large quantities of bile. Adequate drainage by this method can be obtained only by prolonged intubation or by frequently repeated passage of the duodenal tube. Either procedure is distressing to the sick patient and a burden on the attendants besides removing large quantities of bile which is required for normal gastrointestinal and biliary tract function.

**DRAINAGE BY DIET.** This method is more physiological and equally effective. It has the advantage of being capable of producing frequent or almost continuous biliary drainage without depleting the patient. Frequent feedings, six or eight in twenty-four hours, should consist of



the history is not typical. Their differentiation is discussed in the chapter on the Liver (p. 465).

*Pruritus* is usually an accompaniment of obstructive jaundice, but its presence or severity is not indicative of the cause or the location of the biliary tract obstruction. It also occurs at times in hepatitis, but not in hemolytic jaundice.

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So far as the biliary tract is concerned, a complete and careful x-ray study will usually clear up the diagnosis, although failure of filling of the gallbladder may be a troublesome finding, as mentioned under cholecystography. It is evident that only after a careful history and complete study of all suspicious regions can a definite diagnosis be made and the patient assured of adequate care.

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## Gallbladder Diet

- Breakfast** Milk 1 glass  
Cereal 4 to 8 ounces with cream and sugar  
Egg 1 or 2 soft boiled or poached  
Bread or toast and butter  
Fruit raw any kind
- Luncheon** Milk 1 glass  
Egg 1 soft boiled or poached or cheese preferably cream cheese (2- cake)  
Vegetables cooked all kinds  
Salad with dressing of corn or peanut oil and lemon juice or vinegar  
Bread and butter  
Pudding Jello fruit or ice cream
- Supper** Same as luncheon
- Between meals and at bedtime** (and in the night if awake) 1 heaping teaspoonful of powdered gelatin mixed with water followed by 1 glass of milk and if desired, crackers bread and butter or cake  
Vitamin and mineral capsules once or twice daily
- NOTE** In the case of food allergies this diet must be modified accordingly (see p. 91)  
Latent diabetes must be controlled with insulin and avoidance of excessive fat

The caloric value of this diet should be varied according to requirement. Patients may be caused to gain or lose weight by adding to or reducing the quantities of carbohydrates but the fat content must be maintained. We have found by x-ray study that the between meal milk and gelatin feedings stimulate gallbladder emptying almost as well as mixtures containing more fat such as cream (see Fig. 101).

It will be noted that no meat or meat extracts are included in this otherwise varied diet. As the liver is frequently involved in such conditions

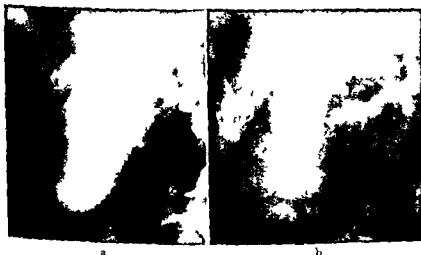


Figure 101 a Dye filled gallbladder large b Dye filled gallbladder 1 hour after milk gelatin mixture. Smaller and more dense

proper proportions of all food elements to provide adequate nutriment. Each feeding should be planned to produce relaxation of the sphincter of Oddi and emptying of the gallbladder and ducts. Fats and oils are important but rarely need to be taken to excess. They are readily supplied in the form of cream, milk, egg yolk and oils. Peptones are provided by easily digestible proteins such as egg albumin, gelatin and milk proteins.

Even when a patient is being treated for *acute symptoms* with vomiting and pain, it is advantageous to produce biliary drainage and reduce common duct pressure. In such cases these foods can be given in the form of a liquid diet similar to the diet used after gastric hemorrhage. The formula is as follows:

### Formula for Gelatin Milk Feedings

FOOD	AMOUNT	CARBOHYDRATE	PROTEIN	FAT	CALORIES
Gelatin	50 gm		45 gm		150
Ceetrose	60 gm	60 gm			240
Dream (40%)	100 cc	6 gm	6 gm	36 gm	360
Milk	900 cc	36 gm	27 gm	27 gm	550
		105 gm	81 gm	63 gm	1330 per liter

In patients allergic to milk this mixture may cause severe symptoms including pain, vomiting or diarrhea. In such cases a milk substitute such as soy bean emulsion and extra fat in the form of a vegetable oil will need to be used.

This mixture may either be prepared at once in 1 liter bottles by first dissolving the gelatin in a little of the warmed mixture and then adding to the full amount, or the powdered gelatin can be kept aside and added to each feeding. If desired a little flavoring of vanilla, cocoa or tea may be added, making the mixture more palatable, although most patients prefer it without. The patient is given 6 to 8 ounces of the formula at two to three hourly intervals about six or eight times a day. Three times daily a raw egg may be beaten into the preparation and mixed vitamin concentrates should be added to it or taken separately. As acute symptoms subside, additions of vegetables and fruit are made to the diet until the regular gallbladder diet is being taken.

In the ordinary *chronic case* I have for over thirty years given the following diet which embodies the principles in regard to fat and peptones and in addition provides an adequate well balanced diet with slightly high protein content:

**Gallbladder Diet**

- Breakfast** Milk 1 glass  
 Cereal 4 to 8 ounces with cream and sugar  
 Egg 1 or 2 soft boiled or poached  
 Bread or toast and butter  
 Fruit raw any kind
- Luncheon** Milk 1 glass  
 Egg 1 soft boiled or poached or cheese preferably cream cheese (1/4 cake)  
 Vegetables cooked all kinds  
 Salad with dressing of corn or peanut oil and lemon juice or vinegar  
 Bread and butter  
 Pudding Jello Fruit or ice cream
- Supper** Same as luncheon
- Between meals and at bedtime (and in the night if awake)* 1 heaping teaspoonful of powdered gelatin mixed with water followed by 1 glass of milk and if desired crackers bread and butter or cake  
 Vitamin and mineral capsules once or twice daily
- NOTE** In the case of food allergies this diet must be modified accordingly (see p. 94)  
 Coexistent diabetes must be controlled with insulin and avoidance of excessive fat

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Figure 101 a Dye filled gallbladder large b Dye filled gallbladder 16 hour after milk gelatin mixture Smaller and more dense

it is important to avoid these substances which as Mann and Bollman long ago demonstrated act as poisons to a damaged liver (see p 461). After symptoms have subsided and the patient has been apparently in good health with no signs of liver damage over a period of six or eight weeks meats and fish can be gradually but cautiously added to the diet.

The effects of this diet may vary. In some patients especially in those whose gallbladders are distended owing to fasting or to a previous fat free diet—as for instance after an attack of biliary colic—the diet may in the beginning occasion considerable distress or even may precipitate an attack of colic. Persistence in the feedings will usually cause a subsidence of symptoms in a week or ten days even in patients with stones. In more than half of patients the diet will relieve the retrostaltic symptoms promptly will relieve the local distress within a week or two and will greatly improve the patients general condition. In cases which showed no gallbladder filling or poor function the good effect of the diet can be demonstrated by x ray films showing good filling and improved function. If no improvement occurs and the gallbladder cannot be demonstrated after six or eight weeks on such a diet complications are usually present and surgery may be required.

*Treatment of Each Specific Disease* The treatment of each such disease of the gallbladder and ducts and their complications will be described below. There are no drugs of specific value in any of these diseases. Antibiotics and at times sulfonamides may be of much value in the presence of definite infection. Mild sedation may be used in the early stages of treatment in neurotic patients but should not be continued for long. Antihistaminics or epinephrine is of value in controlling allergic reactions. In *biliary colic* the pain may be controlled by morphine or demerol. Although morphine may cause spasms of the sphincter of Oddi this spasm is usually overcome by the feeding of fats and oils as described above. Nitrites are not usually required although at times inhalation of amyl nitrite or a sublingual tablet of nitroglycerin will be of value. Antispasmodics have variable effects and I do not use them. Bile acids bile salts and fatty acid preparations are not required if the patient is on an adequate diet. They may even do harm. In the presence of *icterus* if there is laboratory evidence of a bleeding tendency but especially if operation is being considered adequate doses of vitamins K and C and calcium should be given in addition to the mixed vitamins and minerals suggested under the diet. A vitamin K preparation such as Synkavite can be given orally in the form of 5 mg tablets three times daily or in emergency it may be administered parenterally in doses of 5 to 10 mg. Vitamin C should be given in doses of 0.5 to 1 gm a day and calcium gluconate 5 gm a day. Blood prothrombin and bleeding time should be checked frequently to avoid overstimulation of coagulation especially in patients with thromboses.

### Surgical Treatment

**Indications for Operation** Operations should not be performed unless careful study has disclosed definite indications for them. Such indications for each disease will be discussed in the next section but we may say that in general the following constitute the principal indications: (1) an "acute abdomen" in which there appears to be imminent danger of perforation; (2) a history of recurrent typical biliary colic but only in a patient whose complete study has revealed the presence of gallstones and whose general condition does not contraindicate operation; (3) the finding of no gallbladder shadow on repeated x-ray studies even after six or eight weeks of a suitable gallbladder diet in a suitable patient; (4) the finding of definite complications such as (a) a cystic or common duct obstruction from impacted stone or cicatricial stricture with a palpable enlarged gallbladder; (b) pancreatic or hepatic injury; (c) fistulas involving the biliary tract and other organs; (d) various complications following operations upon the biliary tract.

**Contraindications to Operation** Except in an "acute abdomen" when operation may be necessary as a lifesaving measure certain contraindications must be considered: (1) Cardiac decompensation, recent thrombosis or evidence of advanced arterial disease are in general contraindications to operation. However many patients with such complications are greatly benefited by removal of a gallbladder full of stones. If in such patients symptoms can be entirely relieved without operation it is better not to advocate surgery. (2) Any serious disease elsewhere such as a pulmonary lesion, a badly damaged urinary tract, marked obesity, thyroid or other endocrine disorders should cause hesitancy in advising operation. (3) Age must be considered seriously. Aged patients do not stand these operations well. I consider that a patient who is past sixty years of age should not be operated upon unless symptoms persist after prolonged and adequate diet therapy. (4) Unless a skilful, conscientious, experienced surgeon is at hand it is wise to avoid surgery if at all possible.

**Preparation for Operation** This is important. If possible a month or six weeks course of treatment and diet as outlined above will help to prevent postoperative complications. During this period search for and thorough eradication of all focal infections wherever located should be carried out. Complications in the gastrointestinal tract, liver and pancreas should be carefully treated. Other conditions in the cardiovascular, pulmonary, urinary and nervous systems should be evaluated and cared for. Diabetes should be kept under control and its complications avoided. The possibility of allergic reactions especially to milk must be studied. Blood deficiencies should be corrected. Unless some gastrointestinal condition contraindicates it the gallbladder diet should be given until the evening before operation. As a rule preoperative catharsis is not desirable and enemas tend to upset gastrointestinal motility after operation.

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difficult requiring a careful study not only of the gastrointestinal tract to rule out organic causes of symptoms but also cholangiography to detect stones and narrowing or dilatation of ducts. It also should include such a study of other organs and systems as is recommended for preoperative care.

**Treatment** **SURGICAL CARE** Some conditions such as stones, strictures, dilated cystic duct stump, operative injuries, infections and hernias may require operation. Operation should not, however, be performed until a complete study and adequate preparation have been carried out to avoid further disappointment. Conditions other than those in the biliary tract may also require surgical attention. Thorough eradication of focal infections is also imperative. I have seen extraction of a nonvital tooth result in prompt cessation of symptoms.

**MEDICAL CARE** The same treatment is indicated as has been suggested for biliary tract diseases in general, including diet, hygiene and medication. It would also, of course, include adequate care of any complicating diseases such as peptic ulcers, gastritis and colitis, pulmonary, renal or pelvic disorders and spinal injuries. For pain which remains intractable in spite of adequate medical and surgical care, some clinicians have resorted to splanchnic block at first with Novocain and if this helps with alcohol, although this treatment as well as cordotomy is to be frowned upon.

### PROGNOSIS

*Surgical prognosis* should always be guarded. In addition to the many possible annoying and often serious symptoms after operative treatment outlined above, there is also a considerable list of surgical complications resulting in death or at least in disabling disease. These include shock, myocardial, cerebral or pulmonary infarct, pneumonia, subdiaphragmatic abscess, peritonitis, pyelophlebitis and hepatic coma. It is therefore best to follow the rules given when surgical intervention seems imperative.

*Medical prognosis* is more favorable. Those patients who can be spared the risk of operation and who will follow the complete prophylactic and physiological care I have outlined, will nearly all remain well over the years. Cases have even been reported in which stones seen in early cholangiograms have disappeared in six or eight months. The only patients I have seen with recurrence of symptoms are those who have not continued with the frequent feeding schedule, have neglected focal infections or have failed to attend to concurrent diseases elsewhere.

### Anomalies of the Gallbladder and Ducts

It has been reported that nearly 10 per cent of patients coming to operation or autopsy will show some type of congenital abnormality. Some are only of interest, a few produce difficulties or complications indicating the

need for operation or making it more difficult I shall not take time to describe all anomalies that have been encountered but will mention a few of the more common ones

#### ANOMALIES OF THE GALLBLADDER

The *Phrygian cap* is the most common anomaly. This as the name implies consists in a bulge on the fundus of the gallbladder resembling a cap. It causes no disturbance of function and no symptoms and is therefore only of interest. It calls for no treatment.

*Double gallbladder* may consist of two complete gallbladders of equal or different sizes either lying separately or enclosed within a common fascial coat. Each may have a separate cystic duct or they may have one cystic duct in common. Either or both gallbladders may become diseased and cause confusion. The indications for operation are the same as for gallbladder disease in general.

*Congenital absence of the gallbladder* partial or completely intrahepatic gallbladder aberrant pancreatic tissue in the gallbladder and congenital adhesions have been found on rare occasions.

*Intramural diverticula of the gallbladder* have been described both alone and in conjunction with stones. It has been held that these are really Rokitsky Aschoff sinuses normally found in the gallbladder wall which have become dilated permitting gallbladder content to enter them. They may grow to considerable size. The gallbladder x rays usually postprandial films show areas of density around and separated from the density of the contents with perhaps a few communicating with the gallbladder lumen. Inspissation of their contents with calcification will give the appearance of a necklce around the gallbladder. Inflation with gas will produce the so called emphysema of the gallbladder wall. Operation is usually indicated.

#### ANOMALIES OF THE BILE DUCTS

The bile ducts may show *congenital obstruction* or absence of a lumen they may be long duplicated and malposed they may show cysts or accessory ducts or the vascular distribution may vary and varices occur. These anomalies must be borne in mind by a surgeon operating in this region.

#### Diseases of the Gallbladder and Bile Ducts

The preceding sections devoted to a general discussion of the whole problem of diagnosis and care of diseases of the gallbladder and ducts should be studied before this section which relates to individual diseases. It must be borne in mind that disease of one part of the biliary tract almost invariably affects all other parts. The following discussion will bring out the pertinent points to be considered in each disease.

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### PROGNOSIS

*Surgical prognosis* should always be guarded. In addition to the many possible annoying and often serious symptoms after operative treatment outlined above, there is also a considerable list of surgical complications resulting in death or at least in disabling disease. These include shock, myocardial, cerebral or pulmonary infarct, pneumonia, subdiaphragmatic abscess, peritonitis, pyelophlebitis and hepatic coma. It is therefore best to follow the rules given when surgical intervention seems imperative.

*Medical prognosis* is more favorable. Those patients who can be spared the risk of operation and who will follow the complete prophylactic and physiological care I have outlined will nearly all remain well over the years. Cases have even been reported in which stones seen in early cholangiograms have disappeared in six or eight months. The only patients I have seen with recurrence of symptoms are those who have not continued with the frequent feeding schedule, have neglected focal infections or have failed to attend to concurrent diseases elsewhere.

### Anomalies of the Gallbladder and Ducts

It has been reported that nearly 10 per cent of patients coming to operation or autopsy will show some type of congenital abnormality. Some are only of interest; a few produce difficulties or complications indicating the

radiation may be present together with retrostaltic symptoms. The acute symptoms may be ascribed to "indigestion," "stitch in the side," a "virus infection" or a mild pleurisy. The attack may last only a short time. Mild fever and leukocytosis (eosinophilia in allergic crises) and moderate tenderness in the right upper quadrant may be found. The gallbladder may be palpable. In allergic crises there may be a sudden cessation of symptoms especially after administration of ephedrine or an antihistaminic, or symptoms may subside gradually. A history of allergy may be obtained.

*More severe acute cholecystitis* when accompanying an attack of gall stone colic will cause definite pain in the epigastrium or over a tender usually palpable gallbladder. The pain will usually radiate in the characteristic manner and may be aggravated by deep inspiration. The retrostaltic symptoms are usually severe. The pain may be differentiated from that due simply to passage of a stone by the fact that this pain returns after relief by an analgesic. More or less upper right rectus "muscle guarding" may be present. Fever, leukocytosis and increased sedimentation rate are usual findings. Jaundice, which may be due to obstruction of the common duct by stone, edema or pressure, should be watched for by early bilirubin determinations in blood and urine.

*Acute fulminating cholecystitis* gives the findings of "acute abdominal calamity" and often cannot be differentiated from other abdominal conditions such as peptic ulcer perforation, acute appendicitis or an acute intestinal obstruction. Extra abdominal causes of acute symptoms discussed previously under Acute Conditions of the Abdomen (p. 59) in the general section must also be ruled out. The findings make it difficult to decide whether immediate operation should be performed. Surgical consultation therefore is absolutely indicated. A gangrenous gallbladder may perforate while deliberation goes on.

### X-ray Examination

A scout film may help in differential diagnosis of the condition. It may disclose the presence of opaque calculi, may indicate intestinal obstruction as evidenced by gas distribution, or may demonstrate free air in the peritoneal cavity as a result of perforation of a hollow viscus. Air in the bile ducts indicates perforation into them. In the less acute case or after subsidence of a more severe one, cholecystography may show simply a nonfilling gallbladder or the presence of stones. A gastrointestinal study is always indicated at this time.

### Treatment

*Surgical intervention* is frequently the first form of treatment to be considered. While it is best to avoid operation during an acute episode if possible, the danger of complications such as gangrene, perforation, empyema, local or generalized peritonitis and the possibility that torsion

## CHOLECYSTITIS

## ACUTE CHOLECYSTITIS

Acute cholecystitis is a term applied to various acute disturbances from mere edema due to allergic reactions to severe bacterial infection with suppuration gangrene and perforation and liver damage

## Pathology

The pathologic changes observed in the nonbacterial cases may consist only in edema and congestion of the gallbladder wall and to some extent of the ducts. Such cases may be common but are frequently overlooked. Increasing stasis due to obstruction of the outlet of the gallbladder from stones inspissated bile kinks torsion of the gallbladder or angulation due to adhesions to or contact with adjacent structures may occur. This results in the typical greenish pear shaped gallbladder. With interference of blood supply to such a gallbladder more or less gangrene may result and perforation may occur causing a bile peritonitis. More frequently it is covered and walled off by omentum or liver. Rarely such a gallbladder has been found to perforate into the duodenum or colon.

On the other hand acute inflammation or ulcer of the duodenum or pyloric region especially an ulcer perforating into the gallbladder or ducts may cause severe inflammation. An acute pancreatitis is usually complicated by acute cholecystitis. The biliary stasis resulting from the custom of not feeding patients soon enough after any kind of operation and not allowing them sufficient quantities of food especially of fats is the cause of the occasional occurrence of acute cholecystitis a week or two after operations on other organs. Acute gangrenous cholecystitis occasionally occurs after an unrelated operation.

The role of bacterial infection is still in dispute. Some bacteriologists have reported that in a large proportion of acute gallbladder cases no bacteria were detectable. However in cases showing bacterial infection the changes associated with such severe inflammation will be found. This is called *empyema* of the gallbladder and pus may be present in the gall bladder bile. Abscess formation with acute general peritonitis may occur. In bacteremia of various kinds such as coli Welch and typhoid bacillus or virus infections the gallbladder may become infected. Infection may be spread from contiguous organs. In colon bacillus (*Escherichia coli*) infections a fecal odor will be detectable. A demonstrable degree of hepatitis with perihepatitis is a usual accompaniment of acute cholecystitis.

## Symptoms and Diagnosis

Mild acute nonbacterial cholecystitis which really consists only in an edema and congestion of the gallbladder may go unrecognized. More or less pain in the gallbladder region with or without the usual

radiation may be present together with retrostaltic symptoms. The acute symptoms may be ascribed to "indigestion," "stitch in the side," a "virus infection" or a mild pleurisy. The attack may last only a short time. Mild fever and leukocytosis (eosinophilia in allergic cases) and moderate tenderness in the right upper quadrant may be found. The gallbladder may be palpable. In allergic cases there may be a sudden cessation of symptoms especially after administration of ephedrine or an antihistaminic or symptoms may subside gradually. A history of allergy may be obtained.

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### Treatment

*Surgical intervention* is frequently the first form of treatment to be considered. While it is best to avoid operation during an acute episode if possible, the danger of complications such as gallstone perforation, empyema, local or generalized peritonitis and the possibility that torsion



of the gallbladder may account for the acute symptoms makes the decision a difficult one. With torsion there is usually no fever, leukocytosis or jaundice. The possible development of fistulas, pyelophlebitis, multiple liver abscesses, and septicemia in the severe acute case is also a spur to surgical intervention. On the other hand, the accompanying cholangitis, hepatitis and pancreatitis, jaundice and the possibility of cardiac, renal and other complications combine to make a hasty operation risky. Careful continued observation by the doctor and the surgical consultant is the safest course. When operation has been decided upon, every effort should be made to get the patient into the best possible condition to withstand the shock. Transfusions, intravenous dextrose, protein and electrolytes, especially calcium, are usually indicated. Parenteral vitamins, especially K and C, are of particular importance if any jaundice is detectable or even when not. The operation may, in a very ill patient, consist only in drainage by cholecystostomy, although cholecystectomy at once will obviate the need for that procedure later. Postoperative care has been discussed in the general section (p. 54).

When it has been decided not to operate, there is every reason to promote biliary drainage by the use of the dietary measures described in the general section (p. 552), starting with liquid feedings and adding to these as suggested. When the acute symptoms subside without complications, the patient is entitled to a complete study to indicate what needs to be done to prevent future attacks. Many cases will go on to a chronic disease.

### CHRONIC CHOLECYSTITIS

There is still considerable difference of opinion over whether there is such an entity as simple chronic cholecystitis. This has been largely due to the fact that when this diagnosis has been made and the gallbladder has been surgically removed, frequently no benefit has resulted from the operation. There can be no question, however, that such an entity exists.

#### Pathology

The wall of the gallbladder may be thickened and fibrosed. The gallbladder may be large or may be small and contracted. A contracted gallbladder or a large gallbladder filled with stones usually ceases to function. *Cholesterosis*, the "lipoid" or "strawberry" gallbladder, due to infiltration of the wall of the gallbladder with cholesterol, and the *calcified gallbladder* (Fig. 95, p. 543), in which the wall of the gallbladder is infiltrated with calcium carbonate, are variations of chronic cholecystitis (Fig. 102). These gallbladders also usually do not function. With diminished circulation, with chronic irritation and with poor nutrition, it is to be expected that such a gallbladder is a potential danger from the stand-

point of complications. Adhesions to neighboring organs secondary in infections perihepatitis and liver damage are not infrequent complications.

### Etiology

Chronic cholecystitis may result from repeated or from continuous allergic reactions of the gallbladder wall to various agents such as foods, drugs or bacteria. Such allergic reactions to bacteria or the products of bacterial infections in so-called infective foci may produce chronic organic or functional changes in the gallbladder wall without actual bacterial infection, and may thus cause biliary stasis with formation of gallstones. On the other hand, cultures from gallbladder contents obtained by biliary drainage at operation or autopsy have at times shown pathogenic organisms of various types.

It is debatable whether actual bacterial infection accounts for many cases of so-called chronic cholecystitis. Bile salts have a bacteriostatic action so that when infection does occur it is rarely the result of bacteria entering the gallbladder lumen from the ducts, the so-called ascending or descending infection. Usually the bacteria reach the gallbladder wall through the blood vessels or lymphatics or from infection of contiguous structures. In a large proportion of cases the changes described as chronic



Figure 102. Calcified gallbladder. I \ all of nonfunctioning gallbladder. arrow points to transverse colon, containing barium.

of the gallbladder may account for the acute symptoms makes the decision a difficult one. With torsion there is usually no fever, leukocytosis or jaundice. The possible development of fistulas, pyelophlebitis, multiple liver abscesses and septicemia in the severe acute case is also a spur to surgical intervention. On the other hand, the accompanying cholangitis, hepatitis and pancreatitis, jaundice and the possibility of cardiac, renal and other complications combine to make a hasty operation risky. Careful continued observation by the doctor and the surgical consultant is the safest course. When operation has been decided upon, every effort should be made to get the patient into the best possible condition to withstand the shock. Transfusions, intravenous dextrose, protein and electrolytes, especially calcium, are usually indicated. Parenteral vitamins, especially A and C, are of particular importance if any jaundice is detectable or even when not. The operation may in a very ill patient consist only in drainage by cholecystostomy, although cholecystectomy at once will obviate the need for that procedure later. Postoperative care has been discussed in the general section (p. 54).

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functioning gallbladder which may then be shown to contain stones (see p 551). It may be used as the only treatment. Further discussion of this subject will be undertaken in the following section.

### CHOLELITHIASIS (GALLSTONES)

The formation and types of stones have already been discussed. They are practically always associated with evidence of chronic cholecystitis although it is an interesting observation that when patients with gallstones have been properly treated and have had prolonged and adequate dietary care aiming to establish biliary drainage the gallbladder at operation may appear normal. In such cases a conservative surgeon may be tempted a times merely to remove the stones and conserve the gallbladder. Upon removal such gallbladders usually show minimal microscopic changes.

#### Symptoms and Diagnosis

No symptoms may occur even when a gallbladder is packed with stones. Frequently stones are found in the course of a routine gastrointestinal study at operation for another condition or at autopsy in persons who have never had gallbladder symptoms. They have been found at all ages from infancy to old age. Gallstones are commonly found in women of middle age or older and the adage "fair, fat and forty with indigestion" as an indication of gallstones is still borne out in many cases. The "indigestion" consists in retrostaltic symptoms usually rather mild with air swallowing and belching usually the most prominent manifestations. When the typical colic occurs with its right upper pain and characteristic radiation to the right scapula accompanied by nausea and vomiting it calls attention to the biliary tract as a cause especially if followed by jaundice. If infection follows fever, chills and palpation of a mass in the gallbladder region are found. However as emphasized before the diagnosis in an uncomplicated case can be confusing if not confirmed by a complete careful study to rule out other causes of the symptoms and to determine whether complications are present. The diagnosis is considered in the general discussion on gallbladder disease (p 538).

#### Treatment

**Surgical Care.** There is a feeling that the finding of gallstones is an invariable indication for operation. This is definitely not so. There are many patients whom I have followed up for long periods of time up to twenty years or more who never had any complications as long as they followed the rational care prescribed for them. Although cancer does occur in patients with gallstones it is so rare as to be insignificant as a possible cause of death especially as compared with operative mortality. Therefore frightening the patient into an operation by emphasis on the

cholecystitis are considered to be due to irritation or stasis from any cause but mostly from the presence of gallstones the formation of which has already been discussed (p 538)

### Symptoms and Diagnosis

Chronic cholecystitis may exhibit no symptoms and may be discovered only during the course of a complete x ray study. However as over 80 per cent of cases are associated with and probably caused by gallstones symptoms are usually the result of these stones. Aside from the biliary colic resulting from stones or from inspissated bile occluding the neck of the gallbladder or ducts symptoms may be merely retrostaltic such as fullness heartburn sour eructations and belching. In some cases there may be in addition pains in the epigastrium or right upper quadrant occurring one to three hours after meals suggesting peptic ulcer. These may be due to not uncommon coincidental ulcer or merely to duodenal irritation. In the latter case they last only a few days in contrast to the longer duration and periodicity of ulcer pains.

### Physical Examination

Physical examination is of no definite value in making a diagnosis. Mere tenderness in the gallbladder region may be due to many different causes mentioned before (p 549). Even the feeling of a mass may be deceptive. It is important however to make a complete examination not only to search for complications in the abdomen but also to find etiologic factors in general.

### X ray Examination

X ray study is the greatest aid to diagnosis of the condition. In a gastrointestinal and colon study secondary evidences of gallbladder disease may be demonstrable. Cholecystography will frequently show a nonfunctioning gallbladder. This in itself should not be considered an infallible diagnostic sign since it may be functional as described before (p 542). The presence of stones is good evidence of gallbladder disease. Gallbladder cancer must be ruled out if possible.

### Treatment

*Surgical treatment* should not be given first consideration even when the diagnosis is reasonably certain. Removal of noncalculous gallbladders produces notoriously poor results. A nonfilling gallbladder is therefore not in itself an indication for operation (see p 554). The indications for operation in the presence of gallstones will be discussed under Gallstones.

*Medical care* is important. The type of cure and medication described in the general section may be used to restore normal function to a non

functioning gallbladder which may then be shown to contain stones (see p 561). It may be used as the only treatment. Further discussion of this subject will be undertaken in the following section.

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## CHOLANGITIS

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## ACUTE CHOLANGITIS

## Acute Catarrhal Cholangitis

Acute catarrhal cholangitis was formerly thought to be the cause of so called catarrhal jaundice, which we know now is really acute infectious hepatitis with more or less secondary involvement of the intrahepatic and extrahepatic ducts in the virus infection. If acute catarrhal cholangitis should occur alone, it would produce jaundice and other symptoms of obstruction without showing abnormal findings in liver function tests. Cholecystography is contraindicated. Treatment is that described for gallbladder disease and will usually be successful.

## Acute Suppurative Cholangitis

Acute suppurative cholangitis is a form of cholangitis which occurs when bacteria capable of causing suppuration invade the bile ducts, usually secondary to a pre-existing chronic cholangitis, to gallstones or to other obstructing lesions such as adhesions or neoplasms or to operation. More rarely it may be due to systemic infections as mentioned above. The involved ducts show an intense purulent inflammation, the walls become weak, the lumen dilated resembling abscess. The liver becomes swollen and on section will show multiple small cholangiolytic abscesses with necrosis of the liver parenchyma and more or less perihepatitis. The pancreas may be similarly involved and together with the liver may show the complications seen in abscesses of these organs. Perforation may result in peritonitis or in fistulas to neighboring organs.

*Symptoms.* At the onset symptoms may be mild with increasing jaundice, febrile reaction often with intermittent chills and fever ("Charcot's fever"), hepatic and splenic enlargement and leukocytosis. The



danger of cancer is unjustifiable. In young patients under forty who are in good general condition operation is definitely indicated. After fifty and especially after sixty if the patient's symptoms can be relieved entirely by diet resulting in silent gallstones I believe in avoiding operation. Between forty and fifty operation must be considered. If pains persist usually as a result of pressure from the stone filled gallbladder operation should be performed regardless of age if the patient's general condition permits. During an attack of biliary colic it is better not to operate but to keep up the frequent feedings with adequate fat combined with sufficient sedation to relieve the pain (see p 551). With jaundice it is usually desirable to continue the feedings in the hope that the common duct obstruction is only temporary. If it is shown to be permanent operation preceded by adequate vitamin K and C administration and transfusions is indicated. If no stones are found at operation experience has shown that the results will probably not be good. It is always desirable when possible to remove the gallbladder at operation. It is also important that careful exploration of the ducts for stones or for narrowing due to cicatricial contraction or adhesions be carried out. Some surgeons favor routine cholangiography at the time of operation to aid in their discovery.

**Medical Care** This has been fully discussed in the general section (p 550). It consists of (1) a diet with adequate fat content and frequent feedings (2) attention to or eradication of all possible causes of biliary tract disease especially focal infections and allergy (3) adequate care of all complicating disease in other organs.

### Prognosis

The results of adequate medical care are usually excellent. After some initial discomfort the symptoms are relieved and the patient improves in general and may refuse operation because he feels so well. Repeated cholecystographic studies will in most cases show a progressive improvement in gallbladder function even in the presence of stones. If no improvement takes place and symptoms are annoying and sleep inhibiting the patient is a candidate for surgery. Too frequently after operation especially if preoperative and postoperative care have been neglected there may occur a train of symptoms called collectively the postcholecystectomy syndrome. The nature and treatment of this syndrome are described in the preceding general section (p 550).

### HYDROPS OF THE GALLBLADDER

Hydrops of the gallbladder is discussed under cancer of the head of the pancreas causing common bile duct obstruction. The gallbladder is distended with a clear liquid secretion of the gallbladder mucosa. The symptoms and treatment are described in the chapter on the pancreas (p 608).

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**Symptoms.** At the onset symptoms may be mild with increasing jaundice, febrile reaction often with intermittent chills and fever ("Charcot's fever"), hepatic and splenic enlargement and leukocytosis. The

symptoms being those of liver abscess it is usually difficult even at operation or autopsy to determine which came first the liver abscesses or the cholangitis. In liver abscess resulting from pyelophlebitis jaundice is less common and perforation into a bronchus occurs less frequently than in cholangiolytic abscess. Severe cases go on to rapid dissolution but occasionally a chronic purulent cholangitis may ensue and continue for as long as five years.

**Treatment** At times the disease appears almost hopeless. However with the use of massive doses of antibiotics a gallbladder diet of frequent feedings with adequate fat to promote biliary drainage and general measures to support the patient an occasional patient will recover. Surgical consultation should be sought early since operation may be required to drain the gallbladder or ducts to drain individual liver abscesses or to remove a part of the liver.

**Prognosis** In acute diffuse suppuration death is usual. Less severe cases occasionally improve.

#### CHRONIC CHOLANGITIS

The chronic form of cholangitis is also an accompaniment of the other diseases of the biliary tract occurring mostly as a result of irritation from stasis, stones, adhesions, pressure or contiguous chronic inflammatory changes. It may also result from fistulas between the biliary tract and the alimentary canal either as a result of spontaneous perforation or surgical implantation of the gallbladder or common duct into the stomach or small intestine. It may be caused by injury to the duct at operation. It may remain chronic and mild or may be complicated by recurrent attacks of acute cholangitis. Chronic suppurative cholangitis has already been mentioned. A mild chronic so called latent jaundice has been thought to be caused by a slight narrowing of the lumen of the chronically edematous and thickened hepatic or common ducts but usually a more definite cause of the stasis can be discovered. Calcification of the ducts will produce such narrowing. Obstruction of the intrahepatic ducts called *obliterative cholangiolitis* has been described but is extremely rare and it is almost impossible to differentiate it from hepatitis except at autopsy (see p. 497). Obstruction of the cystic duct in the presence of mild chronic cholecystitis will cause precipitation of calcium carbonate from the gallbladder fluid. This may produce milk of calcium bile or the calcium carbonate may be deposited on stones previously present. Common duct obstruction from whatever cause results in jaundice and the backing up of bile into the biliary tract may cause more or less distention of the ducts and interference with hepatic excretion of bile.

#### Symptoms

The symptoms of chronic cholangitis are produced by the condition of which it is a part. The symptoms of chronic cholangitis hep-

itis pancreatitis the biliary colic of gallstones and the pains of duodenal ulcer may be predominant. As has been mentioned before stones may lie in the cystic or even the common duct causing more or less local irritation but unless they lead to obstruction they may produce no symptoms except the history of previous colic. Even obstruction by stones may cause no pain at first but later symptoms will arise due to the backing up of bile. These are usually retrostaltic symptoms with right upper quadrant distress and of course an increasing jaundice. The jaundice is relieved suddenly as the stone is relaxed and passed on or perforation of the stone will produce the same symptoms as described under gallbladder calculi (p. 565). Symptoms occurring after operation suggest injury to the duct or failure to remove stones from the duct at operation.

### Diagnosis

Chronic cholangitis may be assumed to be present in the various conditions noted above. Visual evidence of cholangitis may be seen in cholangiograms which may disclose dilatation and narrowing of the ducts and at times the presence of calculi. Most frequently however the biliary tract fails to be visualized.

### Treatment

The physiologic treatment that is recommended for gallbladder and bile duct disease in general (p. 551) will usually have an excellent effect. Operation is rarely required unless a stone is impacted at some point in the duct or is perforating through the wall of the duct. Chronic suppurative cholangitis usually calls for operation.

### CHOLANGITIS LENTA

This name has been given to an inflammation associated with bacterial endocarditis. It is thought to be due to infection of the intrahepatic bile ducts by the same organism causing the endocarditis—the *Streptococcus viridans*. No distinctive pathologic lesion has been found.

The symptoms are those of the endocardial infection and of the cholangitis.

Treatment of the cholangitis would be the same as for other biliary tract disease plus treatment of the endocarditis.

## NEOPLASMS OF THE GALLBLADDER AND BILE DUCTS

### BENIGN NEOPLASMS

#### Adenomas

Papillary or polypoid adenomas only rarely occur within the gallbladder but are found occasionally in association with gastric or intestinal adenomas. They are extremely rare in the ducts. They may set



Figure 103 a Gallbladder dye filled with polyp (at arrow) b Gallbladder after fatty meal smaller more dense polyp in same location

up some gallbladder irritation with the production of the symptoms found in cholecystitis. The only means of diagnosis aside from surgical exploration is the x ray. Cholecystograms made with the greatest of care will show one or occasionally more than one area of decreased density which remains fixed at one point of the gallbladder lumen in spite of changes of position and degree of filling (Fig 103). Papillomas are more easily recognized because unlike polyps they are not usually associated with gallstones which mask the characteristic findings. Other benign tumors and cysts have been even more rarely found usually at operation. As malignant degeneration of benign tumors is known to occur operation is always indicated.

### Endometriosis

This anomalous condition occurs occasionally in the intestines, rectum and appendix and has been found in the gallbladder. It is usually mistaken for cholecystitis and is recognized only in the pathological laboratory. This subject is more fully discussed in the section on Endometriosis under Intestinal Neoplasms (p 395).

## MALIGNANT NEOPLASMS

Cancer of the gallbladder as a primary lesion is one of the rarest of all cancers of the digestive tract. It occurs mostly in women of the sixth and seventh decades of life. Cancer of the *extrahepatic bile ducts* occurs one fourth as frequently and is much more often found in men. Secondary involvement from neighboring organs is more common than primary cancer.

## Pathology

Adenocarcinoma is the usual primary growth encountered. Squamous cell papillary, scirrhous and colloid varieties have been described. Neighboring sarcomas or Hodgkins disease may also but rarely involve the gallbladder and ducts. The gallbladder always enlarged in primary cancer may grow large enough to obstruct the common duct. Duct cancer is usually located at the junction of the hepatic ducts or in the common duct causing early obstruction. Spread is by direct extension to neighboring organs through blood vessels and lymphatics. The liver, duodenum, pancreas, omentum and aorta become involved and may soon metastasize to lymph nodes along the spine, mediastinum and even to Virchow's node above the left clavicle. Perihepatitis is a complication. Ascites and death may occur before metastases are recognized. The finding of a Krukenberg tumor in the pelvis may be the first hint of cancer. The gallbladder may perforate causing an abscess and subsequent fistula. With the rare cystic duct obstruction there may be empyema of the gallbladder. With common duct obstruction mistaken for obstruction from stones, biliary cirrhosis may occur before the cause is recognized. Cancer of the lower end of the common duct, ampullary cancer, is the usual cause of duodenal cancer. With spread the complicating lesions usually overshadow the gallbladder symptoms. Hemorrhage into the gallbladder, hydrops, liver abscess, peritonitis, portal thrombosis and pylephlebitis will produce violent symptoms.

## Etiology

Although cancer is usually associated with gallstones, the fact that few of these cases develop cancer casts doubt upon stones as a cause. Adenoma is definitely a precancerous lesion.

## Symptoms

At the onset the only symptoms are those of the initial lesion. Gallbladder polyps will usually cause no symptoms but in the gallstone cases there may be a history of previous colics with or without jaundice. An enlarging gallbladder will usually cause pain which will be more or less constant in contrast to biliary colic. With obstruction of the common

or hepatic ducts either from an intrinsic growth or from pressure or invasion from a gallbladder enlarged by cancer a rapidly deepening jaundice will occur. Pruritus may be severe and retrostaltic symptoms will be persistent. A loss of 20 pounds or more in weight is usual. Cancer should be suspected when gallbladder symptoms occur without any let up in a patient with or without known gallstones. When no diagnosis has been made until extensive spread has occurred the symptoms of the complications will overshadow those of the gallbladder.

### Physical Examination

In the early stage of gallbladder cancer there may be only slight tenderness in the right upper quadrant. Icterus develops early. Later the gallbladder is palpable and may be hard in one third to one half of cases. Later still icterus is evident as is cachexia. The liver becomes palpable ascites supervenes signs of involvements of neighboring organs and of secondary infection become prominent. Distant lymph node involvement indicates an incurable condition.

### Laboratory Findings

The laboratory findings will be of little value until complications develop. The blood will show icterus, anemias and later leukocytosis and increased sedimentation rate. *Fractional gastric analysis* may reveal blood in regurgitated duodenal contents. The repeated finding of blood in the contents removed from the duodenum when no ulcer is found is suggestive of biliary tract cancer. Rarely cancer cells have been found on cytologic study of such bloody duodenal contents. *Luer and pancreatic function tests* may indicate involvement of these organs.

### X-ray Examination

The gallbladder will usually not be visualized by cholecystography but even when the gallbladder is seen its malignancy will not be recognized until suspicion is aroused by the findings of pressure upon or involvement of neighboring organs as seen in a gastrointestinal series and barium enema.

### Differential Diagnosis

Carcinoma of the gallbladder and duct is often difficult to distinguish from simple hepatitis especially in its early stages and hepatitis is a usual complication. Obstructive jaundice from gallstones is the diagnosis frequently made before operation and cancer of the pancreas or ampulla of Vater is often suspected. In doubtful cases peritoneoscopy may be of value in visualizing the region and permitting biopsy. *Punch biopsy* a "stab in the dark" is of questionable value.

### Treatment

*Surgical Treatment* Extirpation is the only possible method of treatment but in most cases so much spread has occurred that even if no metastases are present the extensive operation designed by Whipple and mentioned under Cancer of the Pancreas (p 605) is practically never successful. However, as 50 per cent of patients die within six months without treatment it is worth while in patients who show no metastases to try operation. Palliative operations such as cholecystenterostomy or production of an external fistula from above the growth may prolong life a little. Minor resections and transplants are usually only of palliative value.

*Medical Treatment* Before and after operation treatment should consist mainly in providing adequate nutrition orally or parenterally, the use of transfusions as indicated and adequate sedation to relieve pain and allay anxiety. The pruritus may be relieved by adequate doses of vitamins especially of the B complex and vitamins K and C together with calcium gluconate. Details of medical care are discussed under Gastrointestinal Cancer (p 125) and under Cancer of the Liver (p 526) as well as in the beginning of this chapter.

### Prognosis

The mortality rate is almost 100 per cent at present although at times patients have been cured by radical surgery. Jaundice developing as late as a year after operation though usually due to recurrence of cancer may be caused by cicatricial adhesions. Exploration is therefore worth while if the patient is in fair condition.

### EXTERNAL PRESSURE

External pressure on the ducts or gallbladder or adhesions to them may result from enlargement or neoplastic involvement of any of the neighboring organs the stomach duodenum transverse colon appendix liver and pancreas or from primary or secondary involvement of the peritoneum or omentum. The symptoms are not characteristic unless actual obstruction occurs.

The diagnosis is made from observing the deformities by gastrointestinal x-rays cholecystography and cholangiography (Fig. 104). Operation may be indicated if actual obstruction is present.

### STRUCTURES OF THE BILE DUCTS

Strictures may result from acute or chronic inflammation from neoplastic disease from cicatricial contraction of an ulcerative lesion resulting from trauma as from a stone or from surgical procedures.

The symptoms of stricture have already been discussed. Stricture of



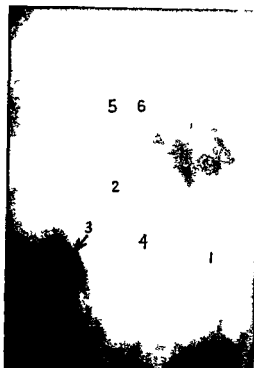


Fig 104



Fig 105

**Figure 104** External pressure on ducts Cholangiogram (tomogram) 1 Common duct 2 cystic duct 3 air in perforated gallbladder 4 recurrent carcinoma after right sided colectomy producing pressure on ducts and gallbladder causing the rounded deformity 5 and 6 right and left hepatic ducts (Used through the courtesy of Lewis L Immerman M D and published in Radiology January 1956 )

**Figure 105** Stricture of common duct following cholecystectomy (cholangiogram) 1 Dilated common duct 2 stricture at middle of common duct (Used through the courtesy of Lewis L Immerman M D )

the cystic duct may cause hydrops of the gallbladder or may result in atrophy or contraction Stricture of the hepatic or common ducts will result in jaundice Strictures may exhibit surprisingly little pain of themselves but will be accompanied or preceded by the symptoms of the conditions causing them Their recognition depends upon an awareness of the possibility that they have occurred on the finding of masses or evidences of distention in their region and on cholecystographic or cholangiographic evidence of obstruction dilatation or stone (Fig 105) Operation is practically always indicated

#### BILIARY FISTULAS

These fistulas are now rarely encountered The wall of a gallbladder packed with stones or one of the bile ducts containing stones imbedded in the wall or prevented from passing by some types of narrowing may perforate spontaneously into the peritoneal cavity into a neighboring

organ or through the skin. At times a stone may cause gradual erosion of increasing depth producing a slow perforation walled off by the nearest organ. Fistulas may also be produced by operations on the biliary tract either as a result of an injury or from a drainage wound which fails to heal. A duodenal ulcer may occasionally perforate into the gallbladder or common duct and produce a similar fistula. The fistula may consist in a communication between the gallbladder or ducts and the stomach, duodenum or colon. In such a case a barium meal or barium enema may be seen to enter the biliary tract or the opaque medium used for cholecystography may be seen to enter the gastrointestinal tract through the tract of the fistula. More rarely biliary fistulas may be found which extend from the gallbladder or ducts to the chest, pleura, bronchial tree or pericardium or to the pelvis, bladder, kidney or large vessels. External biliary fistulas are now rarely encountered because surgeons now rarely drain their gallbladder cases.

### Symptoms

There may or may not be a history of an attack of severe pain at the time of the perforation or patients may fail to recall any episode of such pain. The pain due to a fistula is a typical gallbladder pain and is accompanied by retrostaltic symptoms. Many times fistulas are found during a complete gastrointestinal study instituted because of unusual digestive symptoms. Anemia and loss of weight and strength are not marked except in cases in which an external fistula exists or the loss of much bile which is necessary to digestion.

### Diagnosis

Diagnosis depends upon x-ray study (Fig. 106). In the case of an external fistula cholangiography can be done by injecting an opaque medium into the fistula. At times a mass can be felt in the right upper quadrant.

### Treatment

Internal fistulas practically always require operation preceded by adequate preoperative care much as has been suggested before. In the case of external fistulas which often will heal for months and finally heal spontaneously, it is important to keep the patient in good condition by diet, vitamins and hematinics recommended for biliary tract disease. It is also worth while if possible to reroute the bile to the small intestine. This can be done by collecting the bile as it comes from the external fistulous opening and injecting it into the duodenum through a duodenal tube. When the fistula does not heal it is usually due to obstruction distal to its origin and operation is indicated.

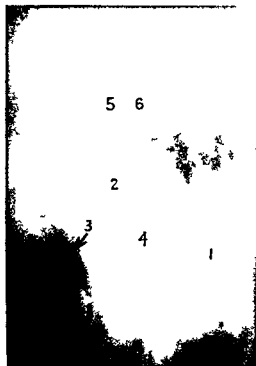


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### Symptoms

There may or may not be a history of an attack of biliary colic at the time of the perforation or patients may fail to recall such pain. The pain due to a fistula is a typical gallbladder pain, usually accompanied by retrostaltic symptoms. Many times there are no digestive symptoms. Anemia and loss of weight are not marked except in cases in which an external fistula drains much bile which is necessary to digestion.

### Diagnosis

Diagnosis depends upon x-ray study. Finding an opaque medium in the right upper quadrant is suggestive of an external fistula. Cholangiography can be done by introducing the opaque medium into the fistula. At times a mass is palpable in the right upper quadrant.

### Treatment

Internal fistulas practically always require adequate preoperative care much as in the case of external fistulas which often heal spontaneously. It is important to treat the patient by diet, vitamins and hematinics. It is also worth while if possible to close the fistula. This can be done by collecting the bile from the fistulous opening and injecting it into the duodenum through a tube. When the fistula does not close, it is distal to its origin and operation is required.

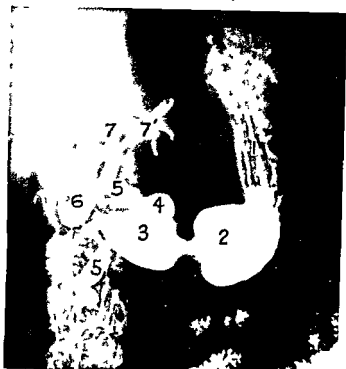


Figure 106 Fistula between duodenum and biliary tract (barium meal study) 1 fundus 2 corpus 3 antrum 4 cap 5 common duct 6 gallbladder 7 right and left hepatic ducts Finer radicles beyond

### Foreign Body

The principal foreign body found in the gallbladder and ducts is the round worm *Ascaris lumbricoides* which originates in the small intestine and may also invade the pancreatic duct and pancreas. Even though these parasites may be destroyed by suitable agents as described in the chapter on Parasitic Diseases (p 147) the dead worm will remain and will produce symptoms of biliary tract disease and obstruction the cause usually not being discovered until operation.

### Trauma

Aside from injury to the gallbladder or ducts trauma may occur at operation. A severe trauma such as from being thrown against the front seat in an automobile accident causing injury to the lower chest or abdomen may produce a rent in the gallbladder or may tear the ducts the cystic artery and often also the liver. The symptoms may consist in acute abdominal pain and vomiting coming on immediately or hours later and calling for immediate operation. If the tear is walled off symptoms of biliary tract disease may follow later. The diagnosis must usually be made from the history and operation will be required.

# The Pancreas

## General Discussion

### ANATOMY OF THE PANCREAS

The pancreas the principal digestive gland of the body is about 12.5 to 15 cm in length of irregular shape and weighs from 60 to 100 gm. It lies transversely across the upper abdomen and is described as having three parts the head body and tail (see Fig. 94 p. 536). Its *head* or largest portion lies in relation to the second and third portions of the duodenum and the pyloric end of the stomach with the liver in front and the vena cava left renal artery and common duct behind. At times the common duct runs through the head. There is no peritoneum posteriorly. The *body* lies largely behind the stomach with the lesser sac between. Posteriorly it lies in contact with the aorta the splenic and renal vessels and the left adrenal. The *tail* starting at the greater curvature of the stomach rests against the lower and inner surface of the spleen. The pancreas resembles the salivary glands but is much softer and looser and has no capsule. It has two ducts the duct of Santorini present in fetal life which may rarely persist and enter the duodenum with or separately from the other the duct of Wirsung. The latter in about 60 per cent of people joins with the common duct and they enter the duodenum through the ampulla of Vater emptying being controlled by the sphincter of Oddi. In the other 35 per cent the duct of Wirsung enters the duodenum separately.

It is important to remember the close relation of the pancreas to its neighboring organs diseases of which may affect the pancreas and in turn pancreatic diseases often involve these organs. It is also of interest to remember that widely differing theories about the etiology of pancreatitis have long been expounded. It was believed for instance that reflux of bile into the pancreatic duct as a result of obstruction or spasm at the ampulla was always the cause of pancreatic disease. Two facts are against this being invariably the case first the fact that the secretory pressure in the duct of Wirsung is much greater than that in the common duct and secondly that the two ducts are not always joined. Pancreatic juice is found not infrequently in the biliary tract as far up as the liver even without obstruction below and causes no symptoms. It is true that bile occasionally enters the pancreatic duct without damage as long as there is no obstruction.

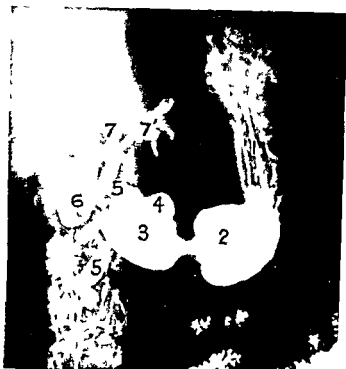


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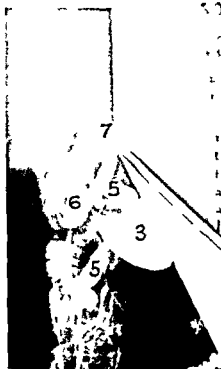


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## SYMPTOMATOLOGY

In early pancreatic disease there are no symptoms except in the case of acute disease. Symptoms are usually caused by complications and include the following:

*Pain* is a prominent symptom and may occur in the upper abdomen, epigastrium and back. It may be referred more to the left than to the right, both anteriorly and posteriorly.

*Retrostaltic symptoms*: epigastric "unrest," fullness, distention, heart burn, sour eructations, regurgitation, belching, nausea and vomiting occur in this as in other gastrointestinal diseases and are not characteristic.

*Steatorrhea* is due to lack of fat digestion and absorption. The stools are bulky, buttery or greasy and foul smelling. Unhydrolyzed neutral fat usually covers the surface like a gray. The more homogeneous greasy stools of malabsorption (sprue) cause diarrhea as a result of irritation from fatty acids and may in addition contain much mucus.

*Malnutrition*: a result of prolonged lack of digestion and absorption, dehydration, loss of weight and other symptoms of food deficiency will occur with advanced disease. Increased appetite is a frequent accompaniment.

In the absence of symptoms patients may at times complain only of the presence of a palpable *mass* in the abdomen.

## EXAMINATIONS

## Physical Examination

In many cases physical signs will not appear until emaciation, dehydration, pallor or jaundice attracts attention. A mass in the upper abdomen, especially to the left, is suspicious of an enlarged pancreas. Secondary enlargement of the liver and spleen may be discovered.

## X-ray Examination

Plain or scout films of the abdomen in different positions may show calcification in the pancreatic region. Osteomalacia of the spine and long bones occurs in advanced disease of the pancreas. The pancreatic shadow has also been studied with artificial pneumoperitoneum and with retroperitoneal air injection as is done in visualizing the kidneys. In a gastrointestinal series following a barium meal, pressure upon or involvement of the stomach and duodenum may be visualized. By means of a barium enema, similar effects on the transverse colon can be brought out. On cholecystographic study, the effect on the gallbladder and ducts is seen. Newer studies include aortography and splenoportography, with films taken immediately after injection of an opaque medium into the aorta or splenic vein respectively. These are done to study the circulation in the portal system. At operation, pancreatography, done by inject

agents on secretion have been studied. It has been pretty well established that pancreatic juice contains aside from enzymes some chlorides and enough sodium bicarbonate to keep the juice alkaline as this is the reaction at which the enzymes do their work.

As with other digestive enzymes it has been found that the normal stimulus to secretion is food. Starvation will reduce the volume of juice to one third. This shows how the postoperative starvation period still inflicted by many surgeons subsequently deranges the digestion of foods and helps to account for the conditions known as "dumping syndrome" and "postcholecystectomy syndrome." The stimulation to secretion is both hormonal and nervous. The secretory hormone *secretin* is formed by the action of hydrochloric acid on the duodenal mucosa. It stimulates the volume of juice. Nervous impulses mainly through the vagus seem to regulate the concentration of the juice. Foods by stimulating gastric secretion with the production of hydrochloric acid provide this acid stimulation to the pancreas. The effect of this stimulation has been shown not to be much influenced by alcohol taken with the food. Secretion is depressed by Banthine and by x-ray a little less by atropine, cocaine and epinephrine. It is stimulated by histamine, pilocarpine, Prostigmin and acetylcholine. The last named drug has been shown to cause vacuolization of the pancreas but this can be prevented by atropine. Morphine causes an increase in serum amylase, codeine and Demerol do not. Relaxation of the sphincter of Oddi can be induced by amyl nitrite which however will not stimulate emptying of the pancreas or gallbladder.

#### PATHOGENESIS

In general pancreatic disorders may be congenital or acquired. The theories as to the part played by the situation of the ducts has been discussed under Anatomy of the Pancreas. The frequent association with pancreatic diseases of biliary tract diseases especially gallstones has been attributed to either cause or effect, some authors holding that gallbladder disease is secondary to that in the pancreas, instead of the older theory that pancreatic disease is due to gallbladder disease. Trauma by causing either rupture or merely contusion to the pancreas often brings on a train of severe symptoms. Malnutrition as in alcoholism especially a deficiency of methionine in the diet results in damage to the acinar cells of the pancreas, with atrophy and fibrosis. Impairment of the blood supply of the pancreas is an important cause. Arteriosclerosis and malignant hypertension not only produce more or less arteriosclerosis in the pancreas but in many instances a ground for areas of focal atrophy and parenchymal necrosis. Acute and chronic infectious diseases notably syphilis, parasitic infestations and diseases in contiguous organs are other causes of pancreatic disease. Benign and malignant tumors are also found.

After three days of this diet the stools are examined microscopically for (1) unhydrolyzed neutral fat which shows up as fat globules either plain or stained red with Sudan III (2) undigested starch granules which are stained blue by an iodine solution and (3) undigested meat fibers recognized by their brownish color and striations. The presence of many such unchanged food substances suggests absence or a great diminution of pancreatic secretion. *Such stool findings are not always present in pancreatic disease.* In sprue and "idiopathic steatorrhea" the fats have been split, so that large numbers of fatty acid crystals can be found.

**Biopsy** Biopsy of the pancreas can be performed only at operation or during peritoneoscopy. Blind aspiration of pancreatic tissue or fluid is not advisable even if the pancreas is large. Punch biopsy of an enlarged liver may only occasionally show cancer metastases or in the case of long standing pancreatic disease a fatty liver will be found.

#### DIAGNOSIS

From the foregoing discussion it can readily be seen why an accurate diagnosis of pancreatic disease is rarely made without operation. No tests are invariably diagnostic, no symptoms are pathognomonic. As has been mentioned, acute pancreatitis is often incorrectly diagnosed although the early elevation of serum enzymes may be of help in creating a suspicion of pancreatic necrosis. Chronic pancreatic disease may be suspected in patients who have recovered without operation from what appeared to be an "acute abdomen" and subsequently have had a fatty "diarrhea" really the passage of bulky soft fatty stools with loss of weight. Pain, especially in the left upper quadrant and back, is another suggestive symptom. The diarrhea of pancreatitis must be differentiated from the diarrheas caused by other conditions mentioned before.

#### TREATMENT

##### General Management

The treatment of pancreatic diseases will be discussed under their respective headings. In general, several principles having a bearing on treatment must be recalled. If secretion is to be *stimulated*, it must be remembered that food is the best stimulant. Secretin, formed by the enterokinase of the duodenal juice plus hydrochloric acid, is also a definite stimulant and several drugs including vagal stimulants such as pilocarpine, Prostigmin, acetylcholine and Mecholyl also belong in this class.

If secretion must be *inhibited*, abstinence from food, removal of gastric juice (to keep hydrochloric acid out of the duodenum) and the use of vagal depressants such as epinephrine, cocaine, atropine and other anticholinergic drugs would be indicated.

**Glucose Tolerance Test** In preparation for the test the patient should be kept on a diet of suitable caloric value containing 300 gm of carbohydrates a day for three days before the test. On the morning of the fourth day after an all night fast the patient receives only 100 gm of glucose (dextrose) in water with the addition of a few drops of lemon juice if desired. Preceding the ingestion of the sugar the blood is examined quantitatively for sugar content and further specimens are removed in one half one two and three hours and even at four and five hour intervals the sugar content being charted as a curve. A normal curve is one showing a fasting sugar level of 80 to 120 mg per 100 ml reaching a high point of 150 to 180 mg at one half hour and gradually falling thereafter to the fasting level at three hours. In diabetes the fasting blood sugar level is high frequently over 170 mg continues to rise to high levels during three hours and is still high at five hours. In hyperinsulinism as in islet cell tumors the sugar starts low even below 60 mg may rise a little rarely to 100 or 120 mg or not at all and will return to the fasting level or lower in two hours. Urine tests for sugar at the same intervals should be negative except in diabetic crises.

Serum Calcium may be low below 8 mg per 100 ml in the fasting state, and may result in tetany. Hypoglycemia gives a bad prognosis as does hypokalemia.

**Stool Examinations** Observations of stools for enzyme content total fats and free and combined fatty acids are accurate only if all foods ingested and the total amount of feces are carefully weighed over several days and subjected to time consuming chemical analyses. In general the bulky fatty milodorous stools of pancreatic enzyme insufficiency contain varying amounts of undigested fats carbohydrates and proteins. It must be realized that a source of error may be the excretion of fat by the duodenal mucosa.

It has been truthfully said that the stool examination after the Schmidt test diet is still better than most tests for pancreatic function. This diet contains neutral fat and partially uncooked meat and starch. A diet embodying these principles is as follows:

#### Diet for Stool Test for Pancreatic Function (Modified Schmidt Diet)

Breakfast	Oatmeal partially cooked 3 ounces
	On soft egg
	On slice of bread with butter (1 inch pat)
	On glass of milk
Lunch	On fried steak 4 to 6 ounces (rare in side)
	On 1 potato (not fully cooked)
	On 1 of bread with butter (1 inch pat)
	On 1 glass of milk
	Jello with whipped cream
inner	Same as lunch
ter	4-5 glass
	medication or other additions to diet

After three days of this diet the stools are examined microscopically for (1) unhydrolyzed neutral fat which shows up as fat globules either plain or stained red with Sudan III (2) undigested starch granules which are stained blue by an iodine solution and (3) undigested meat fibers recognized by their brownish color and striations. The presence of many such unchanged food substances suggests absence or a great diminution of pancreatic secretion. Such stool findings are not always present in pancreatic disease. In sprue and "idiopathic steatorrhea" the fats have been split so that large numbers of fatty acid crystals can be found.

**Biopsy** Biopsy of the pancreas can be performed only at operation or during peritoneoscopy. Blind aspiration of pancreatic tissue or fluid is not advisable even if the pancreas is large. Punch biopsy of an enlarged liver may only occasionally show cancer metastases or in the case of long standing pancreatic disease a fatty liver will be found.

### DIAGNOSIS

From the foregoing discussion it can readily be seen why an accurate diagnosis of pancreatic disease is rarely made without operation. No tests are invariably diagnostic, no symptoms are pathognomonic. As has been mentioned, acute pancreatitis is often incorrectly diagnosed although the early elevation of serum enzymes may be of help in creating a suspicion of pancreatic necrosis. Chronic pancreatic disease may be suspected in patients who have recovered without operation from what appeared to be an acute abdomen and subsequently have had a "fatty diarrhea" really the passage of bulky soft fatty stools with loss of weight. Pain especially in the left upper quadrant and back is another suggestive symptom. The diarrhea of pancreatitis must be differentiated from the diarrheas caused by other conditions mentioned before.

### TREATMENT

#### General Management

The treatment of pancreatic diseases will be discussed under their respective headings. In general several principles having a bearing on treatment must be recalled. If secretion is to be *stimulated* it must be remembered that food is the best stimulant. Secretin formed by the enterokinase of the duodenal juice plus hydrochloric acid is also a definite stimulant and several drugs including vagal stimulants such as pilocarpine, Prostigmin, acetylcholine and Mecholyl also belong in this class.

If secretion must be *inhibited* abstinence from food, removal of gastric juice (to keep hydrochloric acid out of the duodenum) and the use of vagal depressants such as epinephrine, cocaine, atropine and other anticholinergic drugs would be indicated.

**Glucose Tolerance Test** In preparation for the test the patient should be kept on a diet of suitable caloric value containing 300 gm of carbohydrates a day for three days before the test. On the morning of the fourth day after an all night fast the patient receives only 100 gm of glucose (dextrose) in water with the addition of a few drops of lemon juice if desired. Preceding the ingestion of the sugar the blood is examined quantitatively for sugar content and further specimens are removed in one half one two and three hours and even at four and five hour intervals the sugar content being charted is a curve. A normal curve is one showing a fasting sugar level of 80 to 120 mg per 100 ml reaching a high point of 150 to 180 mg at one half hour and gradually falling thereafter to the fasting level at three hours. In diabetes the fasting blood sugar level is high frequently over 170 mg continues to rise to high levels during three hours and is still high at five hours. In hyperinsulinism as in islet cell tumors the sugar starts low even below 60 mg may rise a little rarely to 100 or 120 mg or not at all and will return to the fasting level or lower in two hours. Urine tests for sugar at the same intervals should be negative except in diabetic crises.

Serum Calcium may be low below 8 mg per 100 ml in the fasting state and may result in tetany. *Hypoglycemia* gives a bad prognosis as does *hypokalemia*.

**Stool Examinations** Observations of stools for enzyme content total fats and free and combined fatty acids are accurate only if all foods ingested and the total amount of feces are carefully weighed over several days and subjected to time consuming chemical analyses. In general the bulky fatty milodorous stools of pancreatic enzyme insufficiency contain varying amounts of undigested fats carbohydrates and proteins. It must be realized that a source of error may be the excretion of fat by the duodenal mucosa.

It has been truthfully said that the stool examination after the *Schmidt test diet* is still better than most tests for pancreatic function. This diet contains neutral fat and partially uncooked meat and starch. A diet embodying these principles is as follows:

#### Diet for Stool Test for Pancreatic Function (Modified Schmidt Diet)

- |           |   |
|-----------|---|
| Breakfast | Oatmeal partially cooked 3 ounces<br>One soft egg<br>One slice of bread with butter (1 inch pat)<br>One glass of milk   |
| Lunch     | Chopped steak 4 to 6 ounces (rare inside)<br>Mashed potato (not fully cooked)<br>One slice of bread with butter (1 inch pat)<br>One glass of milk<br>Jello with whipped cream |
| Dinner    | Same as Lunch   |
| After     | 4-5 glass s<br>medication or other additions to diet  |

*Water soluble vitamins* vitamin B complex in full dosages or crude liver injection 2 cc three times a week vitamin C 250 mg a day

Capsules with multivitamins in approximately such doses are available as are solutions for parenteral use

**MINERALS** The most important minerals are *calcium* (in the form of gluconate or other salts) 30 to 60 grains a day and *iron* in the form of ferrous sulfate 5 grain tablets 3 to 6 per day Capsules containing trace minerals are also of value

**LIPOTROPIC AGENTS** Methionine 1 gm choline 2 to 4 gm inositol 0.5 to 1 gm and vitamin B<sub>12</sub> 8 to 10 micrograms orally or parenterally will help to prevent fat deposition in the liver Such combinations are also available

**INSULIN** may be required but must be used with great caution and in the smallest possible dose to prevent hypoglycemia

**HYDROCHLORIC ACID** In the presence of coexisting gastric achylia dilute hydrochloric acid in doses of 1 dram in sugar water should be given one half hour after feedings followed by careful mouth rinsing

Modifications of the diet and additions listed above may need to be made in individual cases but in general the directions as outlined should be of benefit in the average case

### Surgical Treatment

The pancreas has been successfully operated upon therefore in cases in which surgery may be indicated as in malignancy there should be no hesitation about advising operation It is important of course to have the patient prepared for operation if possible by adequate medical care Postoperative care is also important The indications for surgery will be discussed under each disease

### PROGNOSIS

In general the results of treatment medical or surgical as indicated are not outstandingly brilliant but with increasing knowledge of pancreatic physiology and careful observation of postoperative cases constantly better results can be anticipated As an example of enlightened study the following observations have been made after a total pancreatectomy

**Fats** Thirty eight per cent of ingested fat is absorbed after 1.5 gm of pancreatin in twenty four hours 83 per cent after 7.5 gm In some cases patients have gained weight in spite of grossly fatty stools

**Carbohydrate Metabolism** Usually only 20 to 50 units of insulin will be required even on a high carbohydrate diet

**Macrocytic Anemia** This is more apt to occur with complicating cholangitis and will require nutrition therapy and iron

**Fatty infiltration of the liver** may be prevented by the use of phospholipids (lipotropic agents) See above



The pain of pancreatic disease may be so severe that narcotics are not sufficient to relieve it and paravertebral block may be required. It must be borne in mind that morphine raises the blood amylase codeine and Demerol do not.

Antibiotics may be of value in infections of the pancreas and elsewhere.

In chronic pancreatic insufficiency enzymes such as pancreatin and papain are usually required. Vitamins are of great help especially mixed vitamins containing the fat soluble vitamins in water soluble form. Extra methionine is of value and calcium is at times indispensable. In the steatorrhea of pancreatitis emulsifying agents such as Tween 80 are of no benefit. The diet should be balanced but should consist of easily digestible foods. As only the fat absolutely requires the action of pancreatic juice to make it absorbable proteins and carbohydrates need not necessarily be predigested although the proportion lost by lack of complete digestion must be allowed for by giving excessive amounts. A sample diet would be as follows:

#### Diet for Chronic Pancreatic Insufficiency

Calories 3000	Protein 150 gm	Carbohydrates 350-450 gm	Fat 75 gm
Breakfast:	Whole milk 1 glass (8 ounces) Cereal varied (8 ounces) One soft egg Bread 1 slice butter ( $\frac{1}{4}$ of 1 inch cube) Fruit raw (at end of breakfast)		
Mid morning	Gelatin (powdered) 1 heaping teaspoonful mixed or dissolved in water followed by milk 1 glass with dextrose 2 heaping teaspoonfuls		
Lunch	Whole milk 1 glass Cheese 3 ounces or 1 egg Cooked vegetables varied 5 ounces Lettuce large serving with corn or peanut oil and lemon juice Bread 1 slice butter ( $\frac{1}{4}$ of 1 inch cube) Gelatin dessert or stewed fruit		
Mid-afternoon	Same as mid morning		
Supper	Same as lunch		
Water	At least 5-6 glasses a day. No alcohol in any form allowed		
	If diarrhea is excessive some of the bulky vegetables and fruits or the dextrose may be reduced.		
	The caloric value can be raised by adding extra dextrose		
	Additions and substitution may be made as indicated. Meat may be tried after a couple of weeks		

**Supplements to the Diet** PANCREATIN Pancreatic tablets enteric coated 2 or 3 tablets six times daily after feedings. This equals 12 to 18 gm of pancreatin a day. Preparations such as holadin and viokase in similar dosage can be used. Papain was used in the past but is now difficult to obtain expensive and not as good as pancreatin.

**VITAMINS** Fat soluble vitamins (preferably in water soluble form) daily doses of vitamin A 20 000 units of vitamin D 5000 units vitamin K if necessary as shown by prolonged prothrombin time.

**Water soluble vitamins** vitamin B complex in full dosages or crude liver injection 2 cc three times a week vitamin C 250 mg a day

Capsules with multivitamins in approximately such doses are available as are solutions for parenteral use

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<i>Lunch</i>	Whole milk 1 glass Cheese 3 ounces or 1 egg Cooked vegetables varied 5 ounces Lettuce large serving with corn or peanut oil and lemon juice Bread 1 slice butter ( $\frac{1}{2}$ of 1 inch cube) Gelatin dessert or stewed fruit		
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then only after careful microscopic study. X-ray films may show a defect suggesting a neoplasm with or without obstruction. In the duodenum rests may be suspected when peculiar defects are found suggesting polyps. In other cases a rest may have the appearance of a diverticulum. An unusual complication is an islet cell carcinoma in the rest.

### Treatment

In the absence of symptoms or positive findings no treatment is required. If symptoms and the defect or obstruction are found surgery may be required to rule out malignancy. At operation the rests can be removed.

## Functional Disorders of the Pancreas

Although in general nearly all disorders of pancreatic function can be traced to some organic condition that interferes with function, there are some disorders in which no such organic lesion has been found. Both the internal and external secretions may be so affected.

### DISORDERS OF INTERNAL SECRETION

*Hyposecretion of insulin* produces diabetes mellitus. It is a well known fact that typical diabetes mellitus is usually not associated with any demonstrable lesion of the islet cells. So called pancreatic diabetes is associated with chronic pancreatic disease with involvement of or injury to the islet cells.

*Hypersecretion* or hyperinsulinism first described by Seale Harris as a purely functional disease with symptoms of hypoglycemia including the nervous symptoms associated with an insulin reaction has been found so frequently associated with disease of the islet cells particularly adenoma or carcinoma that there is a question whether such involvement has not been overlooked in so called functional hyperinsulinism. This condition will be discussed under Neoplasms of the Pancreas (p. 600).

*Dysinsulinism* is a condition in which symptoms of hyperinsulinism and hypoinsulinism occur alternately apparently is the result of disturbed insulin secretion. These are the patients with diabetes who will at times have spontaneous attacks of hypoglycemia. Such cases require careful diabetic management not within the scope of this book.

### DISORDERS OF EXTERNAL SECRETION

*Pancreatic achylia* has also been described as a functional disorder. It is sometimes in accompaniment of achylia gastrica. I prefer to consider it simply in evidence of pancreatic insufficiency usually due to disease. A pancreatic tumor or chronic pancreatitis with fibrosis or atrophy involving the acini will lower or prevent the secretion of pancreatic juice. The symptoms and signs of pancreatic insufficiency have been discussed

### Pancreatic Anomalies

The *anomalies of the ducts* have already been discussed under Physiology. They consist in the various arrangements of the two pancreatic ducts and the common duct and their relations to the head of the pancreas. Rarely cases of double pancreas with the head and body separate and cases of double tail have been described.

Con<sup>g</sup>enital hypertrophy, atrophy and prolapse have been observed.

#### ANNULAR PANCREAS

This rare anomaly, the head of the pancreas being divided into two arms which wholly or partially surround the duodenum. It may cause no symptoms being found usually only at operation or autopsy. With pressure upon the duodenum as a result of swelling of either the pancreas or the duodenum symptoms suggesting ulcer with obstruction may be present either constantly or intermittently. In acute pancreatitis the obstruction will be acute and will usually be accompanied also by jaundice due to pressure on the common duct.

#### Diagnosis

Diagnosis can be made only on x-ray examination or at operation. Signs of narrowing of the first or second portion of the duodenum may be found occasionally giving the appearance of a diverticulum.

#### Treatment

Various side tracking operations of the duct have been attempted with poor results. These operations as well as attempts at partial resection have resulted in fistulas. The best operations have been found to be those side tracking the duodenum such procedures as duodenojejunostomy, gastroenterostomy and occasionally cholecystenterostomy having been successful.

#### ABERRANT PANCREAS (ACCESSORY PANCREAS AND PANCREATIC RESTS)

Small masses of aberrant pancreatic tissue varying in size from a few millimeters to a few centimeters in diameter may occur in various parts of the gastrointestinal tract and elsewhere even in the mediastinum. In the gastrointestinal tract they are usually submucosal and produce no symptoms unless they grow in size causing irritation or obstruction. In the stomach and duodenum with ulceration in the overlying mucosa there are symptoms and signs like those of ulcer or cancer with hemorrhage as a complication. In the intestines these tissue masses may cause spasms or diarrhea also with mysterious hemorrhages. In the ileum and in a Meckel's diverticulum they may result in an intussusception with bleeding. They are usually identified only at operation or autopsy and

it is almost impossible to decide whether a case may not be one of acute pancreatic necrosis. The rather sudden subsidence of symptoms within a matter of hours or at most a day or two clears up the diagnosis. During the active period however the symptoms and signs are those of an acute necrosis sometimes just as severe. This condition is undoubtedly the initial lesion in necrosis either subsiding spontaneously or going on to necrosis and hemorrhage. When the condition has been caused by a perforated ulcer walled off by the pancreas gastrointestinal x ray studies after subsidence will show an accessory pocket extending downward into the pancreatic area. When it is discovered at the time of a gallbladder operation cholecystectomy usually is all that is required.

#### ACUTE PANCREATIC NECROSIS

Acute pancreatic necrosis is also called acute hemorrhagic pancreatitis or simply acute pancreatitis. The pancreas is usually large friable and dark in color and appears partly digested by its own juices. The mass may be filled with blood. Leakage of its juice produces this autodigestion. Another manifestation is due to free lipase which splits fats in the omentum mesentery and peritoneum. Upon opening the abdomen areas of this fat necrosis are seen scattered throughout the abdomen appearing as small white spots. These consist of calcium combined with the fatty acids produced by the lipase. Large quantities of calcium are used for this purpose so that a lowered serum calcium may be an indication of the severity of the disease. Spilled pancreatic juice also produces some ascitic fluid usually bloody and of course containing large quantities of enzymes. The necrotic pancreas may become infected and suppurative. An acute perihepatitis may be a complication.

#### Symptoms

The onset is violent as a rule and frequently follows a bout of acute alcoholism. The symptoms are generally those of an "acute abdominal calamity" (see p 59) and the diagnosis is usually just that. Occasionally the symptoms may simulate those of coronary or mesenteric thrombosis. The patient is in intense but not colicky pain which may be anywhere in the abdomen and may radiate upward downward or into the back and arms. It is useless to say that a pain in the left epigastrium radiating to the left back is characteristic of pancreatitis. Occasionally it is but the pain may be anywhere. When associated with biliary tract disease or a perihepatitis the pain may be in the right upper quadrant. The patient rapidly goes into collapse with hypotension and tachycardia cold sweat cyanosis and often petechiae or patches of slate blue color on the abdomen and extremities. Obstipation is usual although bloody diarrhea may occur. Vomiting is frequently present and hiccup is a distressing symptom. In the first stage that of pancreatic edema these symptoms may begin to

previously under the general headings of Physiology Symptoms Functional Tests and Treatment for pancreatic diseases in general They will be further discussed under *chronic diseases of the pancreas* (p 595)

### Acute Pancreatic Disease

#### Etiology

The cause of acute pancreatic disease has not been definitely established As mentioned before it has been postulated that regurgitation of bile into the pancreatic ducts is the cause and many cases have been found with a stone impacted at the ampulla of Vater permitting such regurgitation when the pancreatic and common ducts join above this Many cases have been found without stones and with the ducts entering the duodenum separately It has also been pointed out that bile probably enters the pancreatic duct frequently without causing any disease Obstruction of the pancreatic duct could be a factor This and the other possible causes have been discussed under pancreatic diseases in general and include the following

- 1 Biliary tract disease
- 2 Acute infectious diseases including parotitis typhoid fever Weils disease and scarlet fever
- 3 Focal infection anywhere in the body especially following operation on a focus without proper preparation Such foci would include those in the mouth nose and throat pelvis and elsewhere
- 4 Trauma external as from a blow on the abdomen or internal following operations upon the pancreas or neighboring organs such as the stomach duodenum biliary tract or colon
- 5 Poisoning of various kinds including alcohol Chronic alcoholism is a frequent precursor and an acute alcoholic debauch often precedes an attack The pain accompanying methyl alcohol poisoning has usually been found to be due to pancreatic necrosis
- 6 An allergic reaction may produce pancreatic edema which may subside suddenly and unaccountably or may go on to necrosis
- 7 Intestinal parasites which may invade the duct
- 8 Vascular accidents such as embolism apoplexy or thrombosis in the pancreas
- 9 Acute pancreatitis may also be a complication of a pancreatic neoplasm benign or malignant
- 10 A posterior wall duodenal ulcer perforating into the head of the pancreas
- 11 Spasm of the sphincter of Oddi (biliary dyskinesia) has been given as a cause

#### ACUTE PANCREATIC EDEMA

As the name implies acute pancreatic edema consists in acute inflammatory changes with edema The symptoms at onset may be so severe that

calcified pancreas may be seen and its relation to gas shadows may be of help. Gallstones or kidney stones may be revealed. Dilatation of the upper small intestine is rarely seen. The absence of shifting free peritoneal gas in decubital and lateral films rules out perforation of a hollow viscus. A chest film may show abnormalities at the left base: atelectasis, pneumonia, fluid or elevation of the left side of the diaphragm.

### Diagnosis

An accurate diagnosis in 60 per cent of cases is considered good. The conditions usually confused with acute pancreatitis include acute perforations of the stomach, duodenum or appendix, intestinal obstruction and mesenteric or coronary thrombosis. In perforation of a hollow viscus the scout film may disclose free air in the peritoneal cavity. The gas pattern may help in the diagnosis of obstruction. The finding of gallstones may be confusing as between cholecystitis and pancreatitis. The chest findings may be considered as favoring pancreatitis. Electrocardiography may or may not be diagnostic of an acute myocardial infarction, but abnormal findings are common in pancreatic disease.

The following group of findings should create a suspicion of acute pancreatitis: (1) a history of previous similar attacks which subsided without complication; (2) the attack starting after an acute alcoholic debauch; (3) the presence of previous gallbladder symptoms; (4) symptoms of an "acute abdomen" but with only moderate abdominal rigidity and tenderness; (5) changes in the left lung bed; (6) serum amylase over 500 Somogyi units subsiding in a day or two; (7) x-ray films which fail to show evidence of perforation of a hollow viscus.

### Treatment

**Surgery.** Although there is still some difference of opinion as to whether surgery or medical care is indicated, it is a fact that today many surgeons who have opened the abdomen for suspected perforation will close the abdomen without doing anything for pancreatic necrosis if it is present. If gallbladder disease is found to require operation, a cholecystectomy may result in subsidence of the pancreatic disease. With the formerly recommended operations consisting in drainage of the lesser sac, cholecystostomy, choledochostomy or cutting of the sphincter of Oddi, the mortality rate was excessive. Even today with preliminary use of antibiotics and intravenous injections of salt free albumin, it is still well over 20 per cent, whereas under medical management it should be under 10 per cent.

**Medical Care.** Medical care includes several definite objectives, all of equal importance:

1. **Shock** may require norepinephrine, Levophed or corticosteroids as well as care of fluid and electrolyte balance.



subside just as surgical intervention is being considered. When it goes on to necrosis symptoms resembling those of peritonitis occur. At times jaundice will appear usually in the cases associated with biliary tract disease especially with stones complicated by cholangitis. It may also be caused by the pancreas compressing the common duct. In severe necrosis shock may occur but late.

### Physical Examination

The patient often overweight has the anxious worried expression seen with an acute abdomen. holds himself rigid and may stoop over forward. The fever is usually not high between normal and 101° F. The pulse is rapid blood pressure not remarkable. Heart and lungs usually show no abnormalities on physical examination, although in severe cases some changes will be noted at the left base usually due to areas of atelectasis. The abdomen may be moderately or greatly distended. There is usually not much rigidity but some muscle spasm is present. Tenderness varies may be severe and is located in the right upper quadrant at the umbilicus to the left or in the back. Occasionally there is rebound tenderness. Rarely a mass may be palpable in the midabdomen.

### Laboratory Tests

Blood cell counts will show the leukocytosis and elevated sedimentation rate of an acute abdomen occasionally with an elevated eosinophilic count. Blood chemistry shows hyperglycemia in nearly 25 per cent of cases and there may be some glycosuria. Hypoglycemia is mentioned before usually comes late and is a serious finding. There is generally a blood volume deficit and increase in red cell osmotic fragility. The icterus index may be low in the alcoholic type high in that associated with biliary tract disease.

The serum calcium is usually below 9 mg per 100 ml from the second to fifteenth day. When the calcium goes below 7.5 mg it is a bad sign. All patients with less than 7 mg usually die.

Serum enzyme studies are usually of considerable value. An amylase of over 500 units during the first twenty-four hours and later a lipase over 3 or 4 cc or a high antithrombin titer are important although not absolutely certain indications of pancreatic disease as mentioned before. An abnormally low amylase is of serious import indicating hepatocellular disease.

Urine examination may show no specific findings except that in Weil's disease a rare cause of pancreatitis the leptospiral may be found (see under liver diseases p. 527).

### X-ray Examination

This should consist only of flat or scout films. Rarely an enlarged or

## The Pancreas

acute necrosis. However in any of the chronic conditions there are episodes of acute pancreatitis which will require the same study as the acute disease. Fatty liver should be looked for.

### CHRONIC PANCREATITIS

There is considerable difference of opinion among clinicians and pathologists as to just what is meant by chronic pancreatitis. Clinicians of chronic relapsing pancreatitis characterized by recurrent attacks of acute pancreatic necrosis with few or no symptoms between attacks. Pathologists describe a fibrosis resulting from acute necrosis either lobular without involvement of the islets resulting in deficiency of enzyme or interacinar with replacement of parenchyma resulting in severe pancreatic diabetes. These forms may occur separately or together. There is a form in which calcification is the prominent feature. Fatty infiltration is also encountered associated with hemochromatosis or portal cirrhosis. Chronic adhesive perihepatitis may occur. With fibrosis of the head of the pancreas common duct obstruction may ensue causing jaundice.

#### Etiology

The causes of chronic pancreatitis include those which produce the acute form such as biliary tract disease especially cholelithiasis and duodenal ulcer, chronic alcoholism and acute infections elsewhere. Chronic diseases such as portal cirrhosis, hemochromatosis, tuberculosis, syphilis, sclerosis, chronic fibrocystic disease and amebiasis also may cause changes in the pancreas. It has been found in families. Men between thirty and sixty years of age are most frequently affected.

#### Symptoms

Pain is a frequent symptom usually in the upper abdomen on the left side and back but many patients have no pain. In the relapsing form there is a history of previous acute attacks. In most cases symptoms of gallbladder disease including biliary colic have been present in the past and are confusing. In the cases associated with pancreatic lithiasis so-called pancreatic colic may occur resembling biliary colic but with pain more to the left side and to the left shoulder often suggestive of gallbladder pains. These pains may disappear later as the condition "burns its way out." Jaundice is rarely encountered and if found suggests cancer. Diarrhea is a frequent complaint but on careful inquiry and observation of the stools it is found to consist not of liquid stools but of large soft bulky stools (steatorrhea) which heap up in a pan and appear to have a "gravy" over the surface. These large frequent stools are the result of lack of digestion due to the reduction or absence of enzymes. In some cases of interference with secretion of insulin diabetic symptoms will develop so-called pancreatic diabetes. In all well developed cases malnutri-

2 *Loss of fluids and electrolytes* requires replacement by parenteral electrolytes dextrose, and later amino acids and vitamins for support of the patient

3 *Blood loss* may require transfusion of up to 2 pints of whole blood as well as intravenous serum albumin (usually obtained from the Red Cross) 200 ml a day for several days The albumin acts also as a trypsin inhibitor

4 The severe pain may be relieved by Demerol or codeine but at times may require epidural anesthesia or even paravertebral block Pheno barbital is a useful adjunct

5 *Stimulation to pancreatic secretion* by gastric juice entering the duodenum must be prevented by continuous or intermittent gastric suction with nothing given by mouth Secretion is depressed by atropine and other anticholinergics

6 *Spasm at the sphincter of Oddi* interfering with pancreatic and biliary drainage may be relaxed by parenteral administration of nitroglycerin calcium or anticholinergics at regular intervals

7 *Infection* should be prevented or treated by the use of broad spectrum antibiotics preferably combined with Mycostatin to prevent mycotic infection of the bowel

8 *Complications* both medical and surgical should be carefully watched for by frequent examinations Overdosage with albumin may cause a few basal rales albuminuria is to be expected with optimal dosage In diabetic cases only when insulin is definitely indicated should it be used and then in minimal doses under careful observation Hypoglycemia may result in sudden death

After subsidence of these acute symptoms which usually occurs within three to four days the treatments may be gradually discontinued Bland foods without fats may be started by mouth with alcohol to be strictly avoided After a couple of weeks a normal diet with added vitamins and minerals should be prescribed

### Prognosis

Recovery depends upon the amount or concentration of enzymes at the onset of the attack The patients who have had a large meal just before the attack are the ones most apt to succumb Many patients will get well and have no recurrence Some will have recurrences with no symptoms or mild symptoms between attacks Others will be followed by chronic pancreatitis Some will require operations later as in the case of duodenal ulcer perforating into the pancreas Sequelae should be watched for

### Chronic Pancreatic Disease

In the various chronic diseases of the pancreas the symptoms and findings on examination and the treatment differ distinctly from those of

pressure defects. In a study of the small intestines the irritability and the irregular mucosal patterns may be similar to those seen in other causes for food deficiency. The finding of biliary tract abnormalities, especially gallstones, is suggestive of possible pancreatic involvement. At operation injection of the pancreatic duct with an opaque medium may show abnormalities (pancreatography).

### Diagnosis

Many cases of chronic pancreatitis are overlooked because the diagnosis is difficult. In the relapsing cases the history of previous attacks of "acute abdomen" which subsided spontaneously is of help. Pain may or may not be present and is not characteristic. The steatorrhea may often be differentiated from sprue (the malabsorption syndrome) by the finding of neutral fat due to complete failure of the pancreatic enzyme lipase whereas split fats (fatty acids and crystals) are found in sprue. Pancreatic cancer is often difficult to differentiate even at operation and biopsy. Mild diabetes with steatorrhea and a calcified pancreas are definite indications of chronic pancreatitis.

### Treatment

*Prophylaxis* should consist in adequate care, medical and surgical, not only of biliary tract disease but of all of the various conditions mentioned under Etiology. Care of these conditions is also imperative when chronic pancreatitis has developed.

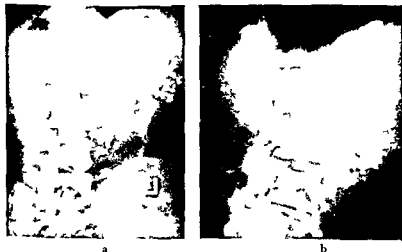


Figure 10—Anteroposterior (a) and lateral (b) views of diffuse calcification of the pancreas associated with multiple pancreatic calcification proved at autopsy in a Negro 51 years of age (Used through the courtesy of Doctors M. C. Sosman and Donovan of the Peter Bent Brigham Hospital.)

a constant finding with weight loss, weakness, languor and occasionally symptoms due to specific food deficiencies. In such cases, especially those associated with calcification, tuberculosis is a common complication in the lungs, intestine and peritoneum. Gastrointestinal hemorrhage may occur.

### Physical Examination

Aside from signs of weight loss, pallor and muscular weakness and more rarely pain, there are usually no specific findings except those of the diseases mentioned under Etiology. Rarely an indefinite tumor may be palpable in the region of the pancreas, usually not moving with respiration. Tenderness is also seldom found, anteriorly over the pancreas posteriorly to the left of the lower dorsal spine. Findings in acute attacks have been described under pancreatic necrosis.

### Laboratory Procedures

In the chronic disease, *blood cell counts* will show an anemia. *Blood chemistries* will reveal hypoproteinemia and in many cases hyperglycemia. Occasionally there may be a transient rise in the *icterus index*. A positive *serological test* should occasion a suspicion that syphilis may be the cause. *Serum amylase and lipase* are usually within normal limits except in acute exacerbations.

*Urine examination* will show glycosuria in cases with islet destruction or the presence of bile in cases with common duct obstruction.

*Fractional gastric analysis* after histamine may show normal findings but hyp acidity or an acidity is frequently found.

*Secretin Test* The finding of diminished amounts of enzymes and a distinctly lowered sodium bicarbonate content are of some significance. An entire absence of enzymes is called "pancreatic achylia."

*Stool Examinations* These are probably most significant. As mentioned before, daily determinations of total fat and protein content of twenty-four hour collections of feces after a test diet are usually not required. Microscopic study of the bulky, greasy stools after a Schmidt test diet (see p. 584) is comparatively simple and of considerable value. The finding of neutral fat globules, plain or stained red with Sudan III and of striated meat fibers and undigested starch granules is good evidence of failure of pancreatic secretion.

### X-ray Examination

Scout films, especially when oblique views are taken, may show calcification (Fig. 107). Rarely a single calculus in the duct of Wirsung may be seen. At times the calcification will be discovered in films of the spine or in cholecystograms taken to discover the cause of pain. In films made after a barium meal the pancreas is usually not large enough to show

pressure defects. In a study of the small intestines the irritability and the irregular mucosal patterns may be similar to those seen in other causes for food deficiency. The finding of biliary tract abnormalities especially gallstones is suggestive of possible pancreatic involvement. At operation injection of the pancreatic duct with an opaque medium may show abnormalities (pancreatography).

### Diagnosis

Many cases of chronic pancreatitis are overlooked because the diagnosis is difficult. In the relapsing cases the history of previous attacks of "acute abdomen" which subsided spontaneously is of help. Pain may or may not be present and is not characteristic. The steatorrhea may often be differentiated from sprue (the malabsorption syndrome) by the finding of neutral fat due to complete failure of the pancreatic enzyme lipase whereas split fats (fatty acids and crystals) are found in sprue. Pancreatic cancer is often difficult to differentiate even at operation and biopsy. Mild diabetes with steatorrhea and a calcified pancreas are definite indications of chronic pancreatitis.

### Treatment

*Prophylaxis* should consist in adequate care medical and surgical not only of biliary tract disease but of all of the various conditions mentioned under Etiology. Care of these conditions is also imperative when chronic pancreatitis has developed.

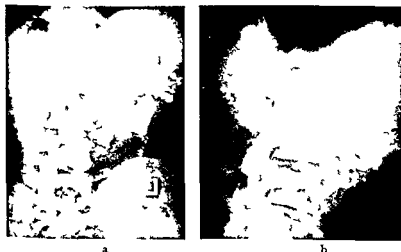


Figure 10—Anteroposterior (a) and lateral (b) views of diffuse calcification of the pancreas associated with multiple pancreatic calculi proved at autopsy in a Negro 51 years of age (Used through the courtesy of Doctors M. C. Sosman and Donovan of the Peter Bent Brigham Hospital.)

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noma fibroma leiomyoma lipoma myoma chondroma and endothelioma. Adenoma is the one usually encountered and occurs as cystadenoma to be discussed under cysts (p. 601), lymphadenoma (rarely) and islet cell adenoma also called nesidioblastoma.

### Pathology

Both the gross and the microscopic findings are those of the tumor pressure upon the functioning pancreatic tissue varying according to the size of the growth. The tendency to malignant degeneration is no greater than with similar tumors elsewhere.

**Islet Cell Tumor.** This is the type most frequently recognized and most prone to undergo carcinomatous change. These tumors vary from 0.1 to 2 cm in diameter, may be single or multiple and occur in any part of the pancreas. They are very vascular, rarely calcified, have no capsule but are not invasive unless they have become malignant. Their cells usually secrete much insulin, causing hypoglycemia, which in time may produce degenerative changes in the cardiovascular and nervous systems. They may go on for years with or without symptoms. When occurring in a diabetic patient there may be alternating attacks of hyperglycemia and hypoglycemia. A diabetic mother may have an infant with this disease, her high blood sugar having stimulated insulin production in the fetus.

### Symptoms

Most of the benign pancreatic tumors will cause no symptoms except in the rare cases in which a tumor may grow to a large size and damage pancreatic tissue or cause pressure symptoms in the abdomen. The islet cell tumor may be recognized if functioning because it produces hyperinsulinism. This does not always occur but when present cannot be differentiated from so-called functional hyperinsulinism. The latter is supposed to be purely an endocrine disturbance associated with disturbances in the adrenals, pituitary, thyroid or thymus. Because of the possibility that it is really due to an islet cell adenoma it should be handled as such. It may undergo malignant change.

The "hyperventilation syndrome" may be associated with the symptoms of islet cell tumor. The chronic symptoms due largely to a moderate degree of hypoglycemia consist mainly in weakness, easy fatigability and hunger before meals, with later symptoms of neurocirculatory asthenia, central nervous system lesions or hepatic cirrhosis. The symptoms of the acute attacks usually those produced by a marked hypersecretion of insulin consist primarily in hunger and weakness with dizziness, trembling, alternating pallor and flushing, sweats and chills, palpitation, salivation, nausea and rarely vomiting. Pain may occur and resemble the hunger pain of peptic ulcer. Nervous symptoms may include fainting, convulsions



*Medical care* includes the measures described for pancreatic insufficiency including (1) a balanced high caloric diet of frequent feedings with added vitamins and minerals (2) pancreatin in large doses 15 to 20 gm a day (panteric tablets 2 or 3, six times daily after feedings) (3) lipotropic agents (methionine choline and inositol) (4) insulin in minimal dosage and only if absolutely required and (5) hydrochloric acid, when a deficiency is found on gastric analysis (6) For the pain simple analgesics or codeine or Demerol may be used Anticholinergic drugs have been tried If severe pain and other symptoms persist, operation may be indicated

*Surgical treatment* requires adequate medical care before and after operation including the use of intravenous salt free albumin as well as blood before and during operation At operation further study by means of direct cholangiography and pancreatography and even biopsy may be done to help in the diagnosis of the lesion to be treated Surgeons disagree as to the proper surgical approach Some do only a cholecystectomy with drainage especially if gallstones are found others only a sphincterotomy to promote pancreatic drainage Anastomosis between the gall bladder or the common duct and the stomach or jejunum have been made as have simple gastrojejunostomies In case of a single calculus in the duct of Wirsung its removal is indicated and calculi may also be removed from other parts of the pancreas In patients with intractable pains epidural and various types of paravertebral block have been performed Rarely has pancreatectomy been resorted to except when there is a grave suspicion of malignancy Removal of the head and body with anastomosis of the tail to the jejunum has been successfully accomplished

### Prognosis

As will be surmised when many medical and surgical methods of treatment have been suggested many cases are difficult to handle None of the operative procedures are a guarantee against recurrence and patients require prolonged medical care thereafter However by means of repeated secretin tests and repeated biopsies it has been shown that complete regeneration of normal pancreatic tissue can occur in some cases under suitable management particularly with complete abstinence from alcohol Alcohol will almost invariably cause recurrences

## Neoplasms of the Pancreas

### BENIGN NEOPLASMS

#### PANCREATIC TUMORS

The pancreas may be involved in many kinds of tumors but they are rare They may occur at any age but are most frequent between the ages of forty and sixty The following benign tumors have been found ade

**Medical Care** Especially in the cases suspected of being functional medical care should consist of a nutritious high caloric diet similar to the one recommended for pancreatic insufficiency (see p 586) but containing large amounts of protein and a smaller proportion of carbohydrates about 150 gm a day of each to minimize insulin stimulation. Vitamins calcium iron and phospholipids should be prescribed as recommended. Overexercise should be avoided. Corticosteroid medication may be tried but should be carefully checked to avoid doing harm. For the acute attack the treatment is the same as for an overdose of insulin. Dextrose by mouth and veins epinephrine or cortisone for shock early feedings with adequate carbohydrate and added dextrose and cautious sedation are indicated. The grave import of the disease should be explained to the patient or his relatives and the indications for sugar at the onset of an attack should be emphasized.

After pancreaticotomy the indications are those previously described for pancreatic insufficiency.

### Prognosis

When single benign pancreatic tumors have been removed the prognosis is good. Medical care for the hyperinsulinism may keep a patient comfortable for years. In the severer cases with recurrent attacks and in those in which a suspicion of malignancy has resulted in a pancreaticotomy fairly good results have been reported when the patients were adequately treated afterward.

### PANCREATIC CYSTS

Cysts are rarely found in the pancreas. They occur in the tail more frequently than elsewhere. They may be congenital. Cystadenomas may grow large up to 100 cc in size may proliferate and may undergo malignant degeneration. They are really tumors. Retention cysts single or multiple vary from a few cubic centimeters to 1000 cc or more in size. Cysts also may occur as a result of degenerative changes and infection with sugar fermentation producing gas cysts or "abscesses." Sebaceous dermoid and so called pseudocysts have also been described. Cysts due to parasites may be found including hidatids and those due to tape worms and roundworms. Fibrocystic disease will be discussed separately (see p 609). Chylous ascites may occur with cysts.

The most common cysts are those resulting from trauma. The trauma may be (1) external due to a flat blunt object such as a steering wheel the cause in more than half of cases. (2) internal following acute pancreatitis or an operation. (3) pressure as from tumors glands an enlarged gallbladder or parasites. On the other hand pressure from a cyst may obstruct the common duct causing jaundice. Cysts occur almost equally in the sexes females showing a slight preponderance.

and colonic spasms with exaggerated reflexes and a positive Babinski sign. Psychiatric symptoms vary from irritability, depression and fear to hysteria, *mania* or unconsciousness so that many patients have been treated for neuropsychiatric disorders. There may be hypertension.

The patient realizing the importance of sugar in relieving an attack usually takes sweets to excess and gains much weight, often becoming obese.

### Examinations

The results of *physical examination* are usually negative since pancreatic tumors rarely grow to palpable size. Pancreatic enzyme tests generally show no abnormalities. In islet cell adenoma the blood sugar findings are definite. Between attacks the *fasting blood sugar* is usually low, under 100, even without symptoms. The oral glucose tolerance test is of little value unless the patient has been on a diet containing 250 gm. of carbohydrate for three days. It then shows a low, almost flat curve, the blood sugar starting at under 100, rarely going over 120 in the second and third specimens and dropping rapidly to low values, staying there for five hours or more. *Intravenously injected glucose* disappears from the blood rapidly.

X-ray films rarely show any abnormal findings unless a large tumor is present.

### Diagnosis

A solid tumor of the pancreas is not recognized unless it is of large size and then it would be practically impossible to differentiate it from a malignant neoplasm except for the usual absence of cachexia. The islet cell tumor is suspected when a patient previously in good health gets weak, nervous and excessively hungry and shows a low blood sugar on a normal diet, lower after fasting or exercise (down to 50 or below). Relief of symptoms by ingestion of sugar and an almost flat sugar tolerance curve are frequent findings. In the more advanced cases the neurological, psychological, cardiovascular and hepatic symptoms should call for study of the sugar metabolism.

### Treatment

**Surgical Care.** The benign tumors, when found and causing symptoms, can be successfully enucleated if single. The islet cell tumors, if large and single, may be found at times and removed. Multiple adenomas amounting to an adenomatosis could be eliminated only by pancreatectomy. This operation has been recommended because of the possibility of malignant degeneration. Occasionally the adenoma may turn out to be in an aberrant pancreas which might be removable. After operation there may be recurrences, malignant changes or fistula formation.

Pentoneoscopy is not advisable exploratory operation being more satisfactory

### Diagnosis

The development of a large abdominal tumor soon after blunt trauma to the abdomen or after an acute pancreatitis or after operation upon or near the pancreas is suggestive of cyst formation. X-ray films may be a help in diagnosis and laboratory findings may be of value in discovering some of the specific types of cysts. It is often difficult to differentiate a pancreatic cyst from other cysts which may occur in the liver, the omentum, spleen, kidneys, adrenals or ovaries and from retroperitoneal tumors, hydronephrosis, dilated gallbladder and even abdominal aneurysm.

### Treatment

**Surgical Treatment** Smaller pancreatic cysts, especially cystadenomas, may be successfully enucleated. For older, larger cysts, marsupialization has been done, but closure of the wound may take six months or more. The operation at present considered most suitable consists in internal drainage by anastomosing the cyst to the stomach, the jejunum or the gallbladder.

**Medical Care** This should be the same as for chronic pancreatitis before and after operation (see Chronic Pancreatitis, p. 597).

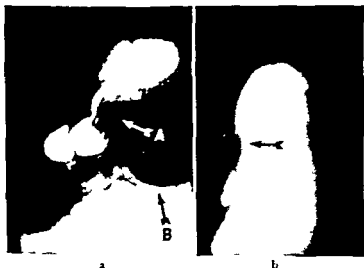


Figure 105. Pancreatic cyst. *a* Arteriogram view. A pressure defect is shown on the curvature of the stomach at arrow A. Note the displacement of the duodenojejunal angle with a pressure defect of the first segment of jejunum at arrow B. *b* Left lateral position showing an anterior displacement of the stomach. (Courtesy of Dr. S. Brown.) (Feldman: Clinical Roentgenology of the Digestive Tract, 2nd ed. Williams & Wilkins.)

### Symptoms

Traumatic cysts are usually associated with pains in the upper part of the abdomen and back. In other cysts the pain is dependent upon the size being due to pressure and occurring usually in the epigastrium radiating to the left and back. The pain is not affected by eating but is relieved by stooping forward. Pressure on the stomach and duodenum may cause retrostaltic symptoms such as anorexia, epigastric discomfort, heartburn, sour eructations, belching, nausea and vomiting. Pressure may also irritate biliary tract or renal symptoms and if the circulatory system is involved ascites may occur. Jaundice may be caused by pressure on the common duct. A large cyst interfering with food intake may result in malnutrition, weight loss and constipation. Some patients report to the doctor because of abdominal enlargement or the feeling of a tumor. Though most cysts tend to grow steadily if left alone patients have been known to live as long as twenty years with a cyst and finally have pancreatic insufficiency. Others may experience sudden acute symptoms due to hemorrhage into a cyst or rupture of a cyst into the bowel or peritoneum.

### Physical Examination

There may be no general findings on physical examination unless nutrition has been affected and the patient appears pale and emaciated. The abdomen, aside from tenderness over the cyst, may show nothing until the cyst has enlarged to the point of causing protrusion. A cyst is usually globular and firm and if large may show fluctuation. There may be transmitted aortic pulsation. Only cysts of the tail will move with respiration.

### Laboratory Findings

Aside from anemia the laboratory findings depend upon complications or upon the extent of involvement. If secondary, acute or chronic pancreatitis occurs the findings of these will be obtained. Parasites or ova may be identified in stools.

### X-ray Examination

Fluoroscopy and scout films of the abdomen may show a large globular tumor with displacement of gas shadows in the stomach and intestine and perhaps diminished excursion of the diaphragm. In the gastrointestinal series and barium enema pressure effects on the stomach, duodenum and colon may be observed (Fig 108). On cholecystography the gallbladder may not be visualized or may fail to empty normally as a result of stasis. At operation pancreatography may show up small cysts.

Peritoneoscopy is not advisable exploratory operation being more satisfactory

### Diagnosis

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### Treatment

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*Medical Care* This should be the same as for chronic pancreatitis before and after operation (see Chronic Pancreatitis p 597)

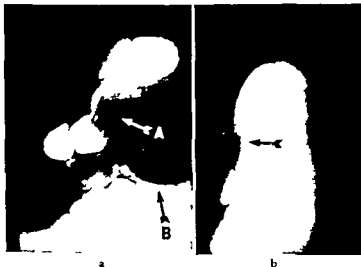


Figure 108 Pancreatic cyst *a* Anteroposterior view A pressure defect is shown on the curvature of the stomach at arrow A. Note the displacement of the duodenojejunal angle with a pressure defect of the first segment of jejunum at arrow B *b* Left lateral position showing an anterior displacement of the stomach. (Courtesy of Dr S Brown.) (Feldman, Clinical Roentgenology of the Digestive Tract, 3rd ed. Williams & Wilkins.)

**Prognosis**

After internal drainage of cysts 80 per cent or more of patients are cured. There is however always the possibility of recurrences. Before and even after operation there is the danger of secondary infection and malignant degeneration.

**MALIGNANT NEOPLASMS**

The pancreas is rarely involved with cancer probably in not more than 5 per cent of all incidence of the disease.

**Pathology**

The lesion is usually a carcinoma. Sarcoma and Hodgkins disease are seldom found and present similar symptoms. Primary involvement is generally either an adenocarcinoma of the parenchyma or much more rarely a carcinoma of the islands of Langerhans called islet cell carcinoma. Rarely a squamous cell carcinoma arises from the ducts and a cystadenocarcinoma from cysts. The head is usually involved the tail and body much less frequently. Secondly the pancreas is invaded by ampullary carcinoma in 10 per cent of cases but in most instances by primary cancer of the liver or stomach. Primary cancer grows fairly rapidly and gradually involves the biliary tract duodenum stomach jejunum and colon kidney and ureters. Metastases are mainly to the liver and lymph nodes. Perihepatitis may be a complication. Pancreatic cancer occurs mostly in patients after fifty years of age and three times as frequently in males as in females.

**Etiology**

The theory has long been held that the primary cancer is due to irritation from reflux of bile through the pancreatic duct as a result of partial obstruction or spasm at the sphincter of Oddi. This theory is being questioned since it has been shown that such reflux of bile occurs in normal persons much more frequently than does cancer. A chronic pancreatitis or pancreatic adenomas or cysts may be precursors.

**Symptoms**

Pain occurs in over 70 per cent of cases. It is usually located in the epigastrium in the right upper quadrant and occasionally in the back. The anterior pain may be relieved by stooping forward the back pain may be increased when the patient lies on his back. It is often an early symptom but is usually attributed by the patient and doctor to other causes. Jaundice is generally a later symptom and is not always present. Weight loss 10 pounds or more may occur fairly early in over 75 per cent of cases but pain practically always precedes it. Most of these

symptoms apply to cancer of the head of the pancreas but all except jaundice occur also in body and tail cancer. Pain may be more to the left in cancer of the body and tail. Anorexia, nausea, vomiting and diarrhea are not characteristic except that the frequent stools may be fatty and bulky owing to pancreatitis accompanying the growth. In nearly one third of cases there is thromboembolism or thrombophlebitis of the femoral or iliac arteries ascribed to a low antithrombin level. Frequently there is a history of previous attacks of biliary colic and the finding of gallstones. The jaundice is attributed to a stone obstructing the common duct. Patients have been operated upon for gallstones and a small cancer of the head has been overlooked because of the frequent association of a hard pancreas due to accompanying pancreatitis. When the stomach or duodenum becomes involved symptoms of peptic ulcer may occur and with gastrointestinal hemorrhage such a diagnosis is frequently considered. With ureteral involvement urinary symptoms including hematuria may become prominent and may be the first symptoms. With colonic invasion cramps and bloody diarrhea may suggest ulcerative colitis. At times cancer of the tail may go on to chest involvement with pleuritis causing symptoms or perforation into a bronchus causing purulent or bloody expectoration. In *islet cell carcinoma* all the foregoing symptoms may occur but as a rule the symptoms are merely those of islet cell tumor. Hypoglycemia is usually present in the early stages of this disease causing hunger pains which are relieved by food especially sugar. Symptoms of an insulin reaction are frequently present.

### Physical Examination

There may be no abnormal findings although emaciation and anemia may suggest malignancy. Icterus usually precedes a palpable tumor. However a tumor may never be felt. With the patient lying on one side the hand may be passed under the ribs and frequently reach the spine behind the stomach where a tumor can be palpated. This should be tried on both sides. The distended gallbladder can frequently be palpated. Metastases to the liver and elsewhere may be the first noticeable signs. The finding of "mocha patches" in the skin is suggestive. Distinct lymphatic involvement is an indication of incurability.

### Laboratory Examinations

**Blood Tests** In the blood anemia and evidences of the icterus (high icterus index, positive direct Van den Bergh) are important. Later evidences of necrosis or secondary infection may be found (leukocytosis, high sedimentation rate). Blood amylase and lipase are so variable as to be of little significance although elevation is suggestive of pancreatic necrosis. In early islet cell cancer hypoglycemia may occur as a result of hypersecretion, hyperglycemia when the islets are destroyed. This





Figure 109 Cancer head of pancreas. Arrows show pressure on and involvement of stomach and duodenum causing widening of the duodenal loop

type of cancer is usually in the body of the pancreas and is accompanied by mental and neurological disturbances. The symptoms are described under islet cell adenoma (p. 599).

**Other Tests** *Fractional gastric analysis* shows no abnormal findings except that blood may be present in regurgitated duodenal contents owing to bleeding from a duct cancer or involvement of the stomach wall by the cancer.

*Duodenal contents* may also contain blood. The secretin test may not show impaired pancreatic function until extensive involvement or severe pancreatitis occurs. Cytologic study of duodenal and gastric content may reveal cancer cells.

*Liver function tests* will often fail to be positive even with considerable involvement of the liver unless acute necrosis supervenes.

*Peritoneoscopy* will frequently show the lesions and permit of biopsy.

*Punch biopsy* is too dangerous except with much liver involvement and even then may be inconclusive.

### X-ray Examination

Diagnosis by x-ray is extremely difficult and requires expert and pains taking study after a barium meal. It depends on the recognition at first

of pressure defects and later of actual involvement of the stomach and duodenum

Cancer of the *head* of the pancreas will produce a gradual widening of the duodenal loop (Fig 109) This is not a constant finding and the widening may also occur in other conditions The greater curvature of the antrum and the duodenum will often show pressure and narrowing or actual involvement At times only irritability or changes in the mucosa

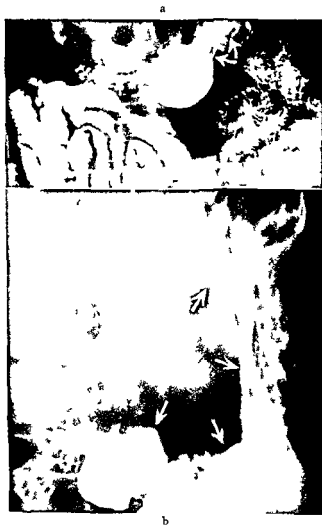


Figure 110 a Cancer tail of pancreas Arrows show involvement of gastric greater curvature high up b Cancer body of pancreas Gastric lesser curvature pressure from large liver with metastases No definite evidence of pancreatic pressure

may arouse suspicion. Delayed duodenal motility or puddling may occur. It may require films in various positions to bring out these findings which may not appear until late. Films taken in the lateral position both standing and lying down may help to reveal a widening of the retrogastric space that is the distance from spine to stomach or duodenum is increased by the mass. This may be further brought out by injecting air into the retroperitoneal space. A barium enema study may disclose pressure or involvement and even perforation of the transverse colon. Cholecystography may show merely a nonfunctioning gallbladder or the presence of calculi.

Cancer of the *body* and *tail* is rarely diagnosed by x ray. Extremely careful study of the upper greater curvature of the stomach may disclose a defect especially in the supine position when the stomach is bent over the tumor (Fig. 110). At times a lesser curvature defect may be seen as a result of pressure or involvement by the pancreas or by liver metastases. Calcifications in the pancreatic area are rarely seen in cancer.

When the growth has invaded the stomach, duodenum or colon or has perforated into the chest cavity, these conditions can be recognized. When the second and third portions of the duodenum are involved it is important to differentiate such involvement from an intrinsic primary carcinoma.

### Diagnosis

Because of its insidious growth pancreatic cancer is rarely recognized early enough to permit of cure. The old adage that painless jaundice means cancer of the pancreas has been proved incorrect. Behavior disturbances due to the lesion may be diagnosed as psychoses or hysteria. Only careful study will make the diagnosis. Frequently the diagnosis is made only at operation or autopsy.

### Treatment

*Surgical Treatment* The only possible cure for any cancer is complete extirpation of all malignant tissue as well as metastases. Formerly pancreatic cancer was considered inoperable and therefore incurable. At first attempts were made to remove involved parts of the pancreas with no success and often with the development of fistulas. It has been found that the palliative by-passing operation of anastomosis of the gallbladder or bile duct to the stomach or jejunum will not prolong life but will give some comfort the patient usually living not longer than six or eight months. With the aid of newer techniques and massive transfusion Whipple designed the operation which includes removal of the pancreas and duodenum and frequently also involved lymph nodes, the stomach, gallbladder, spleen and even the transverse colon. Various anastomoses

are necessary to maintain gastrointestinal function. The mortality rate of this operation has been reduced to 20 or 30 per cent in expert hands and to even less with intravenous injection of 300 to 500 cc. of salt free albumin a day for three to five days. Complications in neighboring organs or the chest may require operation even though no radical cure is attempted. At times removal of all but the tail and an anastomosis between the tail and jejunum has been done.

**Medical Treatment** It must be recognized that medical care is simply palliative. The patient's pain must be controlled by narcotics and sedatives or by paravertebral blocking. The diet should be such as has been outlined for pancreatic insufficiency. If such insufficiency is severe as it always is of course after radical operation, pancreatin in doses of 15 to 20 gm. a day will be required. Insulin must also be given but usually 25 or 30 units a day are sufficient. Antibiotics may be used to control secondary infections.

### Prognosis

In spite of the best palliative medical or surgical care these patients rarely survive more than six to eight months. With severe jaundice this time is considerably reduced. After radical operation which poses usually an immediate high mortality rate even in the best hands, the average survival time is under a year although a very few cases have shown no recurrences in five years. It is usually worth while to attempt the radical operation unless the findings indicate that it would be impossible to perform.

### Fibrocystic Disease of the Pancreas

Cystic fibrosis, fibrocystic disease or mucoviscidosis is a familial disease characterized by widespread abnormal production of mucus. It is usually congenital, occurring in one of a thousand live births and has been found in 2 per cent of autopsies on children. It starts in the embryonic endodermal tube before its division into the respiratory and gastrointestinal tracts. In infancy its manifestations may consist in (1) meconium ileus, pneumonia and pancreatic fibrosis; (2) Pulmonary complications may predominate with atelectasis and pneumonitis and hardly noticeable bowel symptoms; (3) The sweat will show excessive chlorine content with resulting craving for salt.

Most of the children die young from intercurrent disease, usually pneumonia, but a few will live to early adult life. In such cases the principal findings may include diarrhea or steatorrhea, malnutrition, diabetes—primary or secondary—hyperinsulinism due to islet cell adenoma, evidence of pancreatic involvement, the finding of pancreatic cysts and the pulmonary complications. Calcification of the pancreas is rarely seen.

### Diagnosis

Mucoviscidosis should be suspected in a child with frequent or constant diarrhea and respiratory symptoms. Sudden collapse following sweating, great thirst and vomiting after drinking much water is another suggestive symptom. Lowered or absent trypsin and lipase and hyperglycemia add to the diagnosis. It must be noted that no amylase is normally secreted until three months after birth.

### Treatment

A diet such as recommended for pancreatic insufficiency (p. 586) should be forced together with the vitamins, minerals, pancreatin and other measures mentioned in the recommendations. Antibiotics to prevent or control the secondary infections tend to prolong life. Corticosteroids have been used with some success.

### Lipomatous Pseudodystrophy

Lipomatous pseudodystrophy is another childhood disorder rarely occurring in the adult. There are deposits of fat in different parts of the body and the pancreas may be very much enlarged, often to double the normal size and weight (weight up to 170 gm., normal weight 60 to 80 gm.). Although the fat deposits are extensive the islets are rarely involved and there may or may not be signs of pancreatic insufficiency. There is no specific treatment.

### Other Diseases Affecting the Pancreas

There are other diseases which may affect the pancreas as well as other organs, including tuberculosis, syphilis, sarcoidosis, amyloidosis, xanthomatosis, hemochromatosis and plasmacytosis. All these diseases have their principal manifestations elsewhere. When affecting the pancreas sufficiently, they may produce symptoms similar to those of pancreatitis or neoplasm, which will suggest that the pancreas is involved. The treatment would be that of the primary disease with added care of the pancreatic manifestations.

### Foreign Bodies in the Pancreas

The foreign body most apt to be found in the pancreas is a gallstone which has perforated the common duct and become buried in the head of the pancreas. I have seen one case in which a pin had perforated through the duodenal wall and its point had become imbedded in the head of the pancreas, producing chronic symptoms not easily explainable (see Fig. 52, b, p. 298). When the pin was seen by x-ray film and operation was performed, the pin was found. The patient suffered a long

time thereafter from a chronic pancreatitis. A cyst or abscess might have developed.

### Diagnosis

The symptoms are not characteristic. Usually gallbladder or duodenal disease will be suspected. The stone will not be detectable by x-ray film but an opaque body such as a pin will be easily seen although its exact location will be in doubt. Symptoms and laboratory findings suggesting pancreatic disease may be helpful.

### Treatment

Operation is indicated although it will usually be done for gallstones or for some indefinite disease. A visible opaque foreign body of course is a definite finding indicating operation.

### Prognosis

Though the operation should be attended by a low mortality rate the sequelae may be serious.

## Roundworms

*Ascaris lumbricoides* will at times crawl into the biliary tract from the intestine where they live and have been known to travel up the pancreatic duct and to rest in the pancreas. This should be borne in mind when a patient with ascariasis infestation has symptoms of pancreatic disease and the hard pancreas found at operation should be explored in search for worms.

## Trauma of the Pancreas

Injury to the pancreas occurs in three ways: (1) external trauma with violent deep compression of the abdomen usually when the stomach and intestine are empty. Railroad and automobile accidents are the common causes with steering wheel compression the most frequent. Other causes are kicks, deep punches and falls or other injuries associated with abdominal compression. (2) Penetrating wounds from guns or knives which usually also penetrate neighboring organs. (3) injuries at operation as a result of careless palpation or biopsy of the pancreas or in the course of other operations in the vicinity as on the gallbladder, stomach, duodenum, spleen or right kidney. The most frequent surgical injuries are from operation on the stomach. All these types of injuries may cause damage to other abdominal organs and to the spine either alone or together with trauma to the pancreas.

### Pathology

A crushing injury may cause contusion or rupture of the organ. A

bullet may cause a small penetrating wound a knife a deep laceration. An injury will usually cause hemorrhage which may be encapsulated within the pancreas and escape days later or may escape into the lesser peritoneal sac producing a hematoma. Blood may enter the greater peritoneal cavity producing a peritonitis as is seen in acute pancreatic necrosis not due to injury. At times only a traumatic pancreatitis may occur in severe cases pancreatic necrosis is produced. If the patient survives the sequelae to each of these types of injury may be any of those following acute necrosis such as cysts abscess and chronic pancreatitis.

### Diagnosis

Unless the pancreas has been badly torn so that all the symptoms of an "acute abdomen" are present there may be no symptoms referable to the pancreas for several days. In a case of abdominal injury it is easy to miss pancreatic damage. Gradual leakage of blood and pancreatic juice may after several days produce signs of peritonitis or the settling of the fluid in the right lower abdomen may cause a suspicion of appendicitis. In other cases there may be the development of an epigastric mass due to hematoma in the pancreas or lesser sac. With a severe trauma or when necrosis develops later the symptoms and signs of acute pancreatic necrosis (p 591) will be found although differentiation from injury to other organs or even an acute abdomen from other causes may be difficult. The absence of free air in the peritoneal cavity the high serum enzymes and the glycosuria and hyperglycemia should be helpful in creating a suspicion of trauma to the pancreas although not ruling out other concomitant injuries.

### Treatment

It is usually advisable to have a laparotomy performed. Laceration of the pancreas and of other injured organs may be closed and hemorrhage may be controlled but otherwise the operation should conform to that done for acute necrosis.

### Prognosis

Recovery depends on the severity of the injury but the mortality rate is high probably about 50 per cent. Even when recovery takes place the sequelae mentioned before may be of serious import.

## SECTION V

# General Diseases with Manifestations in the Gastrointestinal Tract



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# Gastrointestinal Tuberculosis

With the continued decrease in the incidence of tuberculosis as a result of better understanding of the disease more efficient prophylaxis and constant improvement and simplification of treatment we have been lulled into a feeling that this disease is no longer of clinical importance. This has been particularly true of its gastrointestinal manifestations. With the recent increase in tuberculosis among older people and with the influx of hordes of underprivileged undernourished and diseased people from our Caribbean possessions into our urban centers we may expect that the "white plague" will again be a problem in this country unless thorough prophylaxis and early recognition and treatment are carefully carried out.

Although tuberculosis is generally considered to be primarily a lung infection with spread to other parts of the body from the lungs it has long been known that the tonsils frequently harbor over long periods of time the tubercle bacillus now called *Mycobacterium tuberculosis* and that this is also but rarely the case with infection of lymphoid tissue in the region of the ileocecal valve. A chronic hyperplastic lesion of the rectum has also been described as primary.

Every part of the alimentary canal can be the site of tuberculous involvement with tubercles and organisms demonstrable in nearly all cases. Aside from the tonsils the lip tongue salivary glands and pharynx may show tuberculous lesions difficult to distinguish from other bacterial infections or ulcers and usually not recognized as such unless the patient is known to be tuberculous. Involvement of mediastinal glands may cause esophageal obstruction. Traction from cicatricial contraction of such glands causes traction diverticula of the esophagus.

Laryngeal and esophageal ulcers have been described but are extremely rare except in advanced cases. Either may be the cause of persistent dysphagia.

## THE STOMACH

Ulcers in the stomach occurring in tuberculous patients have been rarely demonstrated to be tuberculous but simple or even carcinomatous ulcers must be carefully differentiated since the symptoms may be the same and the laboratory and x-ray findings equivocal. At times operation may be the safest treatment.



the abscesses and subsequent fistulas which are such a frequent occurrence in old tuberculosis patients. The differential diagnosis among tuberculous ulcers, cancer, syphilis, lymphogranuloma venereum, amebiasis, bacillary dysentery and ulcerative colitis is discussed under The Intestines (p. 350).

Rarely the lesions may be nodular, the nodules being small, brownish red and covered with fine vegetations. These may break down, resembling lupus. Lesions in this area can be recognized upon rectal examination, proctoscopy, biopsy, cultures made from smears and from the fact that they occur in a tuberculous patient. X-ray films will determine the spread upward.

### THE PERITONEUM

Tuberculous peritonitis occurs in two forms:

1 *Acute miliary tuberculosis* in which the peritoneum is studded with tiny tubercles and there is more or less exudate, often bloody, is frequently secondary to ovarian tuberculosis. The onset is acute with vomiting, general abdominal tenderness and distention and spiking fever. A subacute form with prolonged spiking fever was formerly frequently mistaken for typhoid fever. In the ascitic fluid the tubercle organisms may be demonstrable. Porphyria is present at times. These patients are frequently subjected to operation and the lesion is then easily recognized. No surgical procedure is indicated, but medical care is necessary. Occasionally in mild, latent cases the typical findings are discovered at operation for some other lesion.

2 *Chronic hyperplastic or fibroid peritonitis* in which large tumors suggestive of cancer are found is an extremely rare form. The tumors are caused by extensive plastic exudate and broken down tubercles causing multiple adhesions and deformities or fibroid changes. Gastrointestinal x-ray films may show only the narrowings and deformities. With pneumoperitoneum the masses may be demonstrated. Only supportive treatment is indicated.

### THE LIVER

The liver may be involved in the form of small miliary tubercles or large solitary tubercles. It is usually considered cirrhotic. Cirrhosis is not uncommon in tuberculous patients. The diagnosis is usually made at autopsy, although biopsy may disclose tubercles. Treatment for cirrhosis is indicated. Amyloidosis and sarcoidosis are complications (p. 529).

### THE PANCREAS

The pancreas is rarely involved but may also show miliary or solitary tubercles. As a rule no distinctive symptoms can be made out since this

## THE INTESTINES

The intestines are more frequently invaded

*Primary* intestinal tuberculosis occurs mainly in children and may be due to the human or bovine strain. It is usually associated with peritonitis and must be suspected in persistent diarrhea. In extremely rare instances has primary ileocecal or colonic tuberculosis been demonstrated in the adult usually having been mistaken for regional ileitis, ulcerative colitis or malignant lymphoma.

*Secondarily* the ileum, cecum and appendix are frequently involved. The spread from the primary focus takes place through the lymphatics or blood vessels by autoinoculation from the swallowing of bacteria laden sputa or by direct extension from neighboring structures. Ileocecal tuberculosis must be suspected in a tuberculous patient when right lower abdominal pain and diarrhea are the predominant symptoms. Occasionally these symptoms bring the patient to the doctor and unless a careful chest examination is made the primary pulmonary lesion may be overlooked.

Physical examination may indicate an appendicitis. The x-ray finding of extreme irritability of the ileum and cecum alone is suspicious but with careful technique films may be obtained showing barium in a thickened, deformed ileocecal region and occasionally in a fairly large cecal ulcer. The lesions can easily be mistaken for regional ileocolitis or for cancer. Operation is not ordinarily necessary if the lesion is tuberculous unless there is perforation or marked permanent deformity or obstruction caused by infiltration, cicatrization or adhesions. However if operation is performed and the lesion is found to be definitely tuberculous and not requiring surgery, no harm will have been done. It has long been noted that mere opening of the abdomen is of benefit more especially in peritoneal tuberculosis. Excision has not been a favored treatment because of complications due to spread of the infection.

Invasion of the intestine from a tuberculous peritonitis is a fairly regular occurrence. Direct extension from pelvic or retroperitoneal (psoas) infection may also occur.

## THE RECTUM, ANUS AND PERIANAL REGION

Aside from the rare primary hyperplastic lesion mentioned before tuberculous lesions in this area are practically always secondary to tuberculosis elsewhere. The lesions are most frequently ulcerative consisting either of large shallow discrete ulcers with sharply defined undermined edges and a raw gray uneven slightly elevated base and studded with yellow tubercles or confluent ulceration producing large ulcerated areas. The inflammatory process may burrow under mucosa and submucosa through the muscular layer and into the perirectal tissues causing

the abscesses and subsequent fistulas which are such a frequent occurrence in old tuberculosis patients. The differential diagnosis among tuberculous ulcers, cancer, syphilis, lymphogranuloma venereum, amebiasis, bacillary dysentery and ulcerative colitis is discussed under The Intestines (p. 350).

Rarely the lesions may be nodular, the nodules being small, brownish red and covered with fine vegetations. These may break down, resembling lupus. Lesions in this area can be recognized upon rectal examination, proctoscopy, biopsy, cultures made from smears and from the fact that they occur in a tuberculous patient. X-ray films will determine the spread upward.

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### THE PANCREAS

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occurs in advanced tuberculosis in a patient with other more important lesions

### Treatment

Aside from such procedures as paracentesis in ascites and operations for complications there is no specific treatment for the gastrointestinal manifestations of tuberculosis. The patient should be given a balanced nutritious diet (p 21) modified according to the complications present added vitamins and minerals and any local treatments such as demulcents oil enemas and soothing suppositories (see Treatment in chapter on each organ). In general the patient should be treated as in pulmonary tuberculosis in fact nearly all of them have pulmonary lesions.

It is not wise to discuss here the various conflicting ideas about treatment except to indicate that in a patient with gastrointestinal manifestations so called ambulatory treatment should not be instituted too early. These patients need more rest than the average patient with pulmonary tuberculosis alone. With the steady increase in the operative treatment of pulmonary lesions arises the question of removal of more tuberculous foci in the abdomen but this will probably never be done except for complications. The specific chemotherapeutic agents such as isoniazid para aminosalicylic acid (PAS) streptomycin and viomycin are as effective in gastrointestinal as in pulmonary tuberculosis but new agents may be expected to be used as they appear on the market.

### Prognosis

With the modern treatment of tuberculosis a somewhat better prognosis can be given than was formerly the case. Today many patients are being cured of this disease. There is however the danger of the development of local or systemic mycotic and staphylococcal infections as a result of the antibiotics and chemotherapeutic agents now in general use.

## Gastrointestinal Syphilis

With the ease of diagnosis and the simplification of successful and rapid cure of syphilis also called lues it would ordinarily be expected that this disease would soon be entirely wiped out. The reason why this is not coming to pass is the sexual promiscuity of our younger generation even of young teenagers their carelessness or ignorance of the primary lesion of syphilis and their desire to conceal their indiscretions. This is

resulting in epidemics in different parts of the country and an increasing incidence of untreated cases which may not be discovered until later in the tertiary stage. From some of our island possessions we are also importing many untreated syphilitics who cannot be refused admission because of their American citizenship and who are helping to spread the disease. It is therefore of great importance for the clinician to be constantly on the lookout for syphilitic manifestations and it is still good practice to have serologic tests performed routinely on all patients a practice which in recent years has been too much neglected.

Syphilis like tuberculosis and cancer may affect any part of the alimentary canal.

### Congenital Syphilis

Congenital or hereditary syphilis transmitted from a syphilitic mother to her offspring and avoidable if the mother receives antibiotic treatment during pregnancy is not as common as formerly. The liver is the most common site of congenital syphilis. An infant born with a large liver and spleen often with jaundice should be carefully examined for the other characteristic lesions such as interstitial keratitis, mucosal and bone lesions. Serological tests will usually establish the diagnosis. Nearly all these children are either stillborn or die soon after birth. Even with careful treatment they rarely survive more than a few years.

Late hereditary hepatic syphilis, a rare condition, may not appear until the second decade of life and resembles tertiary syphilis. To make certain that it has not been acquired after birth and is truly a hereditary lesion it is necessary to demonstrate such syphilitic stigmata as Hutchinson's teeth, interstitial keratitis, deafness or infantilism. The pathological changes in the liver are so extensive that the prognosis, even with adequate treatment, is very poor.

### Acquired Syphilis

The *primary stage* or chancre, although usually found on the genital organs, is occasionally found on the lips, tongue, buccal mucosa and particularly in the tonsil area or in the anal or perianal region as a result of abnormal sexual practices. As the spirochete, the *Treponema pallidum*, occurs in the lesions of secondary syphilis both in the mucous patches and to a lesser degree in the skin rash, the infection can also be spread by kissing or contact with the secretions from the mouth, penis or anus or by mere dermal contact with abraded skin. It is therefore not necessarily venereal in origin.

The *second stage* occurs six weeks after the primary stage with mucous patches on the mucous membranes or moist skin areas, an often almost unnoticeable general skin rash and sore throat with some fever. While causing definite symptoms, this stage is often overlooked or not recog-



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nized In the *mouth* the patches are mistaken for canker sores and the sore throat is thought to be a cold In the *esophagus* the patches rarely cause any symptoms In the *stomach* they may cause transient indefinite indigestion In the *intestine* or *anorectal* region while condylomas may occur there are usually no symptoms or only slight irritability of short duration In the *liver* jaundice may be caused and may be mistaken for infectious hepatitis

The *tertiary stage* occurs late in untreated or insufficiently treated cases Unfortunately in recent years more than half of the reported cases of syphilis are classed as "early latent" in which the primary and secondary stages have not been treated Tertiary lesions in the gastrointestinal tract consist principally in an interstitial inflammation with fibrosis or in the form of the gumma a tumor of varying size often mistaken for cancer Gummas may be small and multiple or large and solitary Gastrointestinal symptoms may also be caused by the arterial and central nervous system lesions of tertiary syphilis The gastric crises of tabes dorsalis are frequently not recognized patients being treated for peptic ulcer or gallstones The ataxic anal sphincter with a patulous anus and incontinence is another manifestation Rarely are the spirochetes demonstrable in the tertiary lesions although by means of multiple serial sections Warthin claimed he could always find them

The presence of *tertiary syphilis* in different parts of the gastrointestinal tract presents some special problems as follows

### THE ESOPHAGUS

The *esophagus* is rarely affected but both syphilitic induration and *gummas* are occasionally found They produce the symptoms of esophagitis or esophageal cancer respectively as discussed in the chapter on Esophagus (p 207) Antisyphilitic treatment will usually clear up these lesions although at times some local treatment with bougies may be indicated

### THE STOMACH

The *stomach* is also rarely affected in tertiary syphilis Small areas or the whole stomach may show characteristic syphilitic induration resembling linitis plastica single or multiple gummas may occur resembling cancer and necrosis and ulceration over one of the above lesions or independently due to obliterating endarteritis may be confused with simple ulcer

The *symptoms* are those of either ulcer or cancer They tend to be progressive and with increasing size of the lesion may decrease the lumen of the stomach until less and less food can be ingested without a feeling of distention Actual vomiting may occur with forced feedings beyond a certain quantity The appetite usually remains unimpaired

### Diagnosis

Fractional gastric analysis may show normal findings reflex hypersecretion or in achylia. The only real information obtained from it is the diminishing capacity of the stomach, the rapidity of emptying which is increased in fibrosis with achylia and the finding of blood when ulceration has occurred.

### X Ray Examination

X ray films will show either the induration of the wall and decreased capacity, the circumscribed defects due to gummas or the ulcer crater with considerable induration. Differentiation from cancer or ulcer is accomplished by the serological examination as mentioned and the rapid improvement or actual disappearance of the lesion under anti-luetic therapy (see Fig. 50 p. 294).

### Treatment

An ulcer diet (p. 267) with added vitamins and minerals will usually relieve the symptoms but antisypilitic therapy is necessary to cause a rapid disappearance of the lesions as seen by x ray. When the diagnosis of syphilis has not been made and ulceration has progressed to stenosis or perforation operation will be indicated.

## THE INTESTINES

The small and large intestines are even more rarely involved with tertiary syphilis. Any one or a combination of the usual lesions may occur and may cause symptoms of intestinal irritation such as diarrhea with or without bleeding, or those of intestinal obstruction, pains and vomiting. X ray study may cause a suspicion of enteritis, colitis or cancer. Here also obstruction or perforation may occur when the diagnosis has not been made and adequate treatment instituted. In addition to penicillin the treatment is the same as for the lesions it resembles.

## THE LIVER

The liver as mentioned before may be the seat of interstitial fibrosis or cirrhosis or may show single or multiple gummas resembling primary or metastatic carcinoma. The extent of parenchymal damage depends upon the degree of involvement and its duration. Every patient with either of these findings should have a careful study to rule out syphilis.

The liver damage requires treatment as recommended in the chapter on the Liver (p. 530) with the addition of antisypilitic therapy. Because of rapid regeneration of liver parenchyma the prognosis in tertiary hepatic syphilis is better than in portal cirrhosis and much better than in hereditary syphilis.

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### Moniliasis

This term is used in describing infection by *Candida albicans* a yeast like fungus known also by many other names. The organism is widely prevalent having been found in the mouth gastrointestinal tract or vagina in 20 to 30 per cent of normal persons. It is frequently found in the sputum of patients with tuberculosis or cancer of the lung and in the diarrheal stools of sprue and pernicious anemia. It is held to be the cause of thrush and infections dermatitis in moist skin areas and vaginitis. To it are attributed the proctitis or even colitis with persistent diarrheas often with ulceration which frequently follow therapy with broad spectrum antibiotics (see p 320). Sensitization may occur producing a variety of allergic symptoms. Rarely it causes systemic infections with abscess formation and meningitis producing a high mortality.

The usual lesions are granulomatous or tubercle like. In the colon severe infections or allergic reactions to the fungus may resemble ulcerative colitis.

### Diagnosis

It is often difficult to establish this organism as the cause of specific lesions. Skin and agglutination tests are practically of no value since they may be positive in normal persons. Smears or cultures from the lesions or from scrapings made from the rectal lesions through a proctoscope are of value.

### Treatment

Though formerly moniliasis was difficult to cure it has been found recently that Mycostatin is an effective treatment. It is given by mouth in tablets of 500,000 units from one to three times daily. It can also be used locally. The drug can be given with antibiotics to prevent monilial infection from their use.

### Blastomycosis

Two forms of *Blastomyces* are recognized. *Blastomyces dermatitidis* producing skin and rarely pulmonary lesions is seen only in North America. *Blastomyces brasiliensis* is the cause of South American blastomycosis. It occurs in the form of large thick walled round or oval yeast cells or as budding forms. Endemic in South America it may be found in patients who have visited there. Infection takes place through the mouth extending to adjacent skin areas to the tonsils and thence to lymphoid follicles in the gastrointestinal tract liver and spleen. Ulcerative vegetative papillomatous lesions occur in the mucosa and skin. In the abdomen the involved lymphoid tissue may resemble Hodgkin's disease. Diarrhea often with bleeding is a prominent symptom (see p 313).

chapter and more specifically in the chapters devoted to each part of the gastrointestinal tract

### Actinomycosis

Actinomycosis is due to *Actinomyces bovis* or ray fungus so called because colonies on culture media show a raylike arrangement. Grossly conglomerations of the organisms occur as sulfur granules which can usually be demonstrated in the discharges from lesions. The fungi are anaerobic and normally inhibit mucous membranes of the respiratory and gastrointestinal tracts. Infection occurs as a result of trauma causing a break in the mucosa allowing the organisms to enter the deeper tissues. The trauma may consist in injury from curious teeth or from operations such as extractions or tonsillectomy accounting for the facial and cervical infections. More than half of the cases however have been shown to be due to rupture of some part of the gastrointestinal tract such as perforation of the appendix or peptic ulcer of colonic diverticula or rectal abscesses or of the colon from swallowed sharp objects such as fish bones. The vascular lesions with their marked fibrotic reaction often resemble those of cancer, syphilis, tuberculosis or other chronic infection. Spread of the infection to the liver, lungs and genital tract is not unusual and skin lesions with sinuses draining the internal lesions are common. Secondary infections occur occasionally and further complicate the picture by producing high fever and marked leukocytosis which are usually only of moderate degree.

*Diagnosis* is difficult. The gastrointestinal lesions as has been mentioned may resemble almost any disease due to other causes such as cancer, inflammation or ulceration and these must be ruled out by careful study. In the liver abscess and fibrotic changes may occur usually secondary to ileocecal or lung invasion. Serologic and skin tests are of little if any value. The diagnosis depends on finding massed colonies of the ray fungus (sulfur granules) in exudates from the lesion by cultures or in biopsy material. An occasional finding of a colony in sputum, gastric content or bowel scrapings is not conclusive evidence of infection since they may be found in normal persons.

*Treatment* consists in surgical drainage when abscesses have occurred and in accessible surgical removal of the fibrotic infected areas and the use of massive doses of penicillin or broad spectrum antibiotics over a long period. Penicillin is started in dosage of 1 million units every four hours. A combination of the latter with Mycostatin is valuable to control and prevent monilial infection of the bowel. Increasing doses of iodine in the form of potassium iodide were much used in the past but exert no specific action.

Formerly considered practically a fatal disease actinomycosis now shows a mortality rate as low as 20 per cent.

by the tiny *Histoplasma capsulatum* a yeastlike organism which may invade the reticuloendothelial system and be found in the phagocytic mononuclear blood cells. It may cause ulcerations in the mouth, nose, pharynx and larynx and also may cause pulmonary lesions, the symptoms resembling those of an upper respiratory tract infection, bronchitis or tuberculosis. Milium calcified arcs may result in the lung. Visceral lesions may resemble leishmaniasis. In the abdomen the lesions may resemble malignant lymphoma, Hodgkins disease, leukemia, regional enteritis or ulcerative colitis (see pp. 347 and 352).

### Diagnosis

Stained smears may cause confusion with Leishman-Donovan bodies but cultures are fairly conclusive.

### Treatment

No specific treatment is known. Atabrine may be of value. Many cases subside spontaneously on supportive therapy. The new antimycotic medications may be tried.

### Summary

It is evident that in any abdominal disease in which the symptoms are bizarre and complete study fails to disclose the exact cause of lesions found, especially those resembling neoplastic disease, the possibility of mycotic disease must be borne in mind. In manifestations obviously due to allergy it must be remembered that the mycoses may be the cause.

## Cardiovascular Diseases and Gastrointestinal Diseases

Frequently symptoms of cardiovascular disease are interpreted as being due to gastrointestinal disease and just as frequently the reverse is true. Furthermore, diseases of the two tracts are frequently coexistent. It is often difficult, therefore, to differentiate the symptoms of each and to decide on the suitable treatment.

### The Heart

"Acute indigestion" or gallstone colic and occasionally "acute abdomen" have been diagnosed when the pain of an acute *myocardial infarction*

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*Diagnosis is made by finding the yeast in direct smears or by cultures showing the forms mentioned above*

### Treatment

Full doses of the sulfonamides destroy the organisms

### Coccidioidomycosis

Caused by the *Coccidioides immitis* this disease is also called coccidioidal granuloma San Joaquin fever valley fever and other names. Occurring mostly in wild rodents it has also been found infecting domestic animals. Human infection occurs from inhalation of dust containing spores. No direct transmission from animal to man or man to man has been observed. Infecting the bronchi and lungs it resembles tuberculosis and may also rarely involve lymph nodes skin bones joints larynx meninges and internal organs including the gastrointestinal tract. It is endemic particularly in the San Joaquin valley and parts of Arizona. The granulomatous lesion may remain localized in the lungs and subside spontaneously under symptomatic treatment. The disease may spread rapidly or progressively like miliary tuberculosis to any part of the body causing either granulomatous masses or abscesses proving rapidly fatal. Sensitivity to the fungus may cause allergic reactions anywhere in the body including erythema nodosum and erythema multiforme. Leukocytosis and later lymphocytosis may be present.

### Diagnosis

The rare lesions in the gastrointestinal tract may be mistaken for cancer. A skin test positive to coccidioidin has the same significance as a tuberculin test in tuberculosis. It is found particularly when allergic manifestations occur. Agglutination tests are of limited value. Smears from pus sputum gastric content stools and pleural fluid disclosing the large nonbudding thick walled spores containing multiple endospores make the diagnosis. Cultures show cottony colonies.

### Treatment

There is no specific treatment. The primary form usually subsides spontaneously. The spreading type is often fatal. Rest and nutritious balanced diet with added vitamins and minerals and symptomatic treatment for complications are desirable. The new fungicide Mycostatin in tablets of 500,000 units three times daily may be tried either alone or in combination with broad spectrum antibiotics.

### Histoplasmosis

This is a rare disease occurring in various parts of the world but reported mainly in the United States especially in the South. It is caused

apoplexy may produce a gastric hemorrhage. Sclerotic vessels causing diminished blood supply or actual infarction to the abdominal organs may also cause pains and functional changes of various kinds often attributed to other causes.

**Infarction** of mesenteric blood vessels usually the superior and very rarely the inferior mesenteric artery or veins and their branches or tributaries occurs mostly in men past forty. It may be due to infection of the bowel wall or adjacent disease or may be due to other causes as follows:

**ARTERIAL EMBOLISM** may result from vegetations in acute and subacute bacterial endocarditis from left ventricular mural thrombi with myocardial infarction from thrombi formed in the left atrium in arrhythmias (atrial fibrillation) and from atheromatous plaques from the wall of the aorta.

**ARTERIAL THROMBOSIS** may be due to atherosclerosis to occlusion by a plaque or a piece broken off from hemorrhage under it to obliterating endarteritis Buerger's disease or Raynaud's disease heart failure lupus erythematosus intestinal hypodystrophy and some blood dyscrasias including leukemia splenic anemia and polycythemia vera.

**VEIN THROMBOSIS** of the mesenteric veins is usually secondary to liver disease to phlebotrombophlebitis to congestive heart failure to injury at operation or from strangulated hernia. It may also result from infection in viscera drained by the portal veins such as appendicitis pelvic inflammatory disease and colonic infection or cancer.

### Pathology

The part of the intestine whose blood supply is from the occluded vessel will go through the various stages of dying tissue with successive edema hyperemia ulceration and coagulation necrosis and with perforation and peritonitis as an end result. In such a case blood will be found in the lumen of the gut and in the peritoneal fluid. The condition may occur at any age. It is twice as frequent in males between fifty and seventy years of age as in females. Rarely a minor thrombosis may go on to recovery. Even more extensive occlusion if coming on gradually may result in collateral circulation with survival.

### Symptoms

The onset may be sudden or gradual with increasing mid or lower abdominal pain and symptoms of shock and toxemia. Retrostaltic symptoms are succeeded usually by those of real obstruction. Hyperperistalsis of the lower bowel may or may not be present with diarrheal stools often containing blood.

### Physical Examination

The general findings of shock are more or less evident with fever and tachycardia. The abdomen is distended usually with some rigidity at first.



## 628 General Diseases Affecting Gastrointestinal Tract

is more upper abdominal than precordial and is accompanied by nausea vomiting and shock. Deaths attributed to "acute indigestion" are usually due to such infarction.

Anginal pains are frequently interpreted as heartburn or gastric pain and such patients are treated for ulcer.

Congestive heart failure with its passive congestion of the abdominal viscera enlarged liver and occasional jaundice often goes unrecognized while the patient is treated for gastrointestinal neurosis hepatic cirrhosis or cancer.

Dilatation of the left atrium with compression on the esophagus easily shown by x ray will cause dysphagia. Such patients are often treated for cancer hiatus hernia or cardiospasm. Anomalies of the aortic arch may also cause dysphagia or even erosion of the esophagus.

Acute pericarditis often causes such severe epigastric pain that patients may be mistakenly operated upon for an "acute abdomen." Chronic pericarditis may be associated with adhesions to the esophagus with dysphagia. Pericardial effusion may cause dysphagia.

On the other hand esophageal conditions such as spasm diverticulum cancer and hiatus hernia produce precordial symptoms and are often overlooked while the patient is being treated for supposed heart trouble.

The pains of peptic ulcer of intestinal spasm and of biliary colic are wrongly interpreted as anginal in origin and such patients are often treated for coronary occlusion.

The fainting shock and pallor associated with a gastric hemorrhage are often attributed to a "heart attack" and a history of such attacks is often considered evidence of a "weak heart."

The acute pains and shock of an "acute abdomen" have been often misinterpreted as due to an acute myocardial infarction and patients have been treated for this until a general peritonitis became fatal.

The importance of careful cardiac and gastrointestinal study in patients presenting the symptoms noted must be emphasized. When both lesions occur coincidentally adequate care of the cardiac as well as the gastrointestinal condition is indicated whether the patient requires operation or not.

### Vascular Diseases

Vascular diseases may not only cause symptoms resembling those of a gastrointestinal lesion but also frequently will actually produce organic changes in the gastrointestinal tract.

Arteriosclerosis and hypertension will not only produce many of the cardiac symptoms just mentioned but also may cause atrophy of the gastric mucosa resembling chronic gastritis. Occlusion of a terminal arteriole will cause an arteriosclerotic ulcer similar to an ordinary ulcer and not distinguishable clinically therefrom. Rupture of a vessel or

downward through the diaphragm and produce severe abdominal pains difficult to differentiate from other causes of pain. Rupture of an aneurysm may occur into the esophagus with profuse hematemesis and melena into the mediastinum with terrific pain and cardiac and respiratory embarrassment or in the case of a dissecting aneurysm into the stomach or peritoneal cavity. In any case the symptoms are severe pain, shock and death.

### Physical Examination

Physical examination carefully conducted will demonstrate the upper thoracic pulsating mass with a systolic thrill. Pressure on the subclavian artery by the aneurysm will produce a difference between pulse and blood pressure in the arms. Passage of a stomach tube or bougie in suspected esophageal narrowing may rupture an aneurysm. It is therefore important to rule this out by careful x-ray study first.

### X-ray Examination

Fluoroscopy shows the pulsating mass in the region of the aortic arch. The pressure effect on the esophagus can be shown by a swallow of barium. Films will show the aneurysm. Careful film study of the esophagus is necessary to rule out organic disease.

### Laboratory Procedures

Positive serological tests indicating syphilis together with a history of syphilis make the diagnosis of syphilitic aneurysm. Arteriosclerotic aneurysms are accompanied by hypertension and other signs of the disease.

### Treatment

Aside from antisyphilitic therapy which must be used with great caution because of the danger of rupture due to sudden absorption of the syphilitic induration various operations have been devised. Wiring with great lengths of wire to encourage coagulation has been much used. Wrapping the dilated area with various fabrics has been tried. More recently excision of the aneurysm with replacement of the part of the removed aorta by homografts or by various prostheses has been successful in selected cases.

### ABDOMINAL ANEURYSMS

Aneurysms of the abdominal aorta unlike those of the thoracic aorta are predominantly (85 to 90 per cent) arteriosclerotic in origin. They may be small or large. They occur mainly between the ages of forty and seventy-five, being most prevalent in the sixties and are more frequent in men. Left alone abdominal aneurysms will cause death usually within five or six years. 60 per cent of the patients dying during the first year

## 630 General Diseases Affecting Gastrointestinal Tract

and flaccidity later. Rarely a mass may be palpated in the abdomen or through the rectum.

### Laboratory Findings

The blood cell count will show evidences of infection and necrosis: high neutrophile leukocyte count and increased sedimentation rate.

### X-ray Examination

Scout films will show gas patterns of upper intestinal obstruction: distended midabdominal loops of small intestine above the lesion with no particular changes in the pattern of the colon. In the rare case with colonic involvement the findings will be the same as in colonic obstruction from other causes.

### Diagnosis

A sudden abdominal calamity (see *Acute Conditions of the Abdomen* p. 59) coming on in a patient known to have any of the diseases prone to the development of mesenteric thrombosis is highly suggestive. However, the diagnosis is not established except at operation.

### Treatment

Operation is the treatment with resection of the affected part of the intestine. Anticoagulant therapy may be used but with caution.

### Prognosis

Except in the rare instances mentioned, the patient's life depends upon prompt operation as it does in any other cause of an "acute abdomen." In infants, resection of even small amounts of small intestine is usually fatal. In adults, resection of less than one third or even one half of the small intestine is compatible with life, and of course large segments or the whole colon may be safely removed. The immediate prognosis depends upon the patient's general condition before skillful surgery and adequate preoperative and postoperative care. Prognosis for the future is not very favorable since patients are liable to recurrences in the remaining gut.

## ANEURYSMS

### THORACIC ANEURYSMS

Aneurysms of the thoracic aorta and its branches are usually syphilitic although in recent years more are recognized as being due to other causes, mainly arteriosclerosis. As the aneurysm enlarges it causes increasing pressure upon the esophagus with resulting precordial pain and dysphagia usually mistaken for one of its other causes. With long-standing pressure erosion of the esophagus as well as of the spine may occur with symptoms of esophagitis or ulcer. Dissecting aneurysms may dissect

narrow point and some atrophy beyond and was of further help. This procedure is now used preliminary to excision of the distal aorta below the narrow point and its replacement either by an arterial graft or preferably by a prosthesis made of tightly woven or braided orlon, dacron or other synthetic cloth. The prosthesis can also be made to replace one or both iliac arteries. This prosthesis becomes rapidly converted into a tough fibrous tube with an endothelial lining more satisfactory than the graft and results have been satisfactory. Patients must of course be selected with great care but all are grave risks. However the mortality rate from operation has been reduced to 10 per cent in suitable patients and ten year survivals have been reported. Even a rupture if discovered in time may be wrapped with a cloth like the one used for the prosthesis the cloth becoming incorporated into the aortic wall and stopping the bleeding. The prognosis would therefore not be as bad as formerly.

### Summary

It has been demonstrated that cardiovascular diseases may produce symptoms resembling those of gastrointestinal diseases and that the reverse is often also true. It is therefore important again to emphasize that although a patient may complain only of gastrointestinal symptoms a complete gastrointestinal study important as it is may result in no benefit to the patient because no search was made for other additional causes of symptoms.

## Respiratory Diseases and Gastrointestinal Diseases

Diseases of the respiratory tract are frequently associated with and are even the causes of gastrointestinal symptoms or diseases. The reverse also holds true in many cases.

### Gastrointestinal Conditions

Symptoms or disease of the respiratory tract due to gastrointestinal conditions include the following:

#### ESOPHAGUS

Esophageal conditions of all kinds may produce direct or reflex chest symptoms such as a feeling of compression with difficult breathing often

## 632 General Diseases Affecting Gastrointestinal Tract

after discovery of the aneurysm. In nearly all cases the patients have other arteriosclerotic lesions, those of the coronary arteries being most serious. Occasionally aneurysms may be found elsewhere in the abdomen, as in the splenic or hepatic artery, but particularly in the common iliac arteries. The aorta and the rim of the aneurysm may be calcified. Rupture of the aneurysm causes sudden death, although at times the aneurysm may be covered by surrounding retroperitoneal tissues so that some patients may not have a final rupture for days or even months later. Some aneurysms rupture into neighboring organs. Pressure may cause erosion of the bodies of the vertebra against which they are pressed and in prolonged cases may compress the spinal cord. Fortunately for modern surgical measures most of the abdominal aneurysms are in the terminal aorta below the outlet of the renal arteries.

### Symptoms

Symptoms are variable. Fifteen or 20 per cent may be symptom free until they rupture. Seventy per cent have pain, usually in the epigastrium or low back, but at times in the right or left upper or lower abdomen or left flank. Pressure on surrounding organs may cause distress, pressure on the spinal cord may cause radicular pain. Usually there is anorexia, weight loss and constipation. Rupture may cause sudden death or may produce severe pain, sometimes only in the spine.

### Physical Examination

This will usually show evidence of generalized arteriosclerosis. In over half of the cases a mass may be felt, and if pulsation can be definitely established or a bruit or hum can be heard, the diagnosis is determined. Aside from this the diagnosis is made by x-ray. Direct visualization by fluoroscopy of a pulsating fixed mass is of great help. On plain films a mass may be made out directly, erosion of the spine may be demonstrable and calcium deposits may be clearly seen. Retroperitoneal density will indicate a walled off rupture. A gastrointestinal study following a barium meal may show displacement by the mass of the stomach, intestine or sigmoid. Pneumoperitoneum has been used to localize the mass. Aortography may fail to show the aneurysm because canalization of a thrombus may show a small smooth tube, but this is a suggestive finding. Serological study should be made to find the rare syphilitic aneurysm.

### Treatment

Formerly there was no cure, death being the only prospect. Later wiring, filling the sack with wire injected into it, might delay death for a while. Still later in the distal cases wiring was accompanied by the application of a band, preferably of rubber, around the aorta above the aneurysm, but below the renal arteries. This produced fibrosis at the

narrow point and some atrophy beyond and was of further help. This procedure is now used preliminary to excision of the distal aorta below the narrow point and its replacement either by an arterial graft or preferably by a prosthesis made of tightly woven or braided orlon, dacron or other synthetic cloth. The prosthesis can also be made to replace one or both iliac arteries. This prosthesis becomes rapidly converted into a tough fibrous tube with an endothelial lining more satisfactory than the graft and results have been satisfactory. Patients must of course be selected with great care but all are grave risks. However the mortality rate from operation has been reduced to 10 per cent in suitable patients and ten year survivals have been reported. Even a rupture if discovered in time may be wrapped with a cloth like the one used for the prosthesis the cloth becoming incorporated into the aortic wall and stopping the bleeding. The prognosis would therefore not be as bad as formerly.

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### Gastrointestinal Conditions

Symptoms or disease of the respiratory tract due to gastrointestinal conditions include the following:

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Esophageal conditions of all kinds may produce direct or reflex chest symptoms such as a feeling of compression with difficult breathing often

## 634 General Diseases Affecting Gastrointestinal Tract

with cough *Cancer* of the upper esophagus involving the recurrent laryngeal nerve produces the typical hoarse cough and hoarse voice. Marked obstruction from organic disease and more particularly from cardiospasm frequently allows regurgitated contents to enter the trachea causing irritation cough and bronchitis, occasionally with bronchiectasis. The respiratory symptoms may be so predominant that their real cause may be overlooked.

### STOMACH

Stomach conditions associated with epigastric lower chest and back pains especially when accompanied by aerophagia belching and cough may be overlooked. Belching which may also be associated with esophageal disease may become severe may produce dyspnea and may be mistaken for a cough the so called stomach cough. *Gastric hemorrhage* is often difficult to differentiate from pulmonary hemorrhage. The supposedly typical frothy appearance of the latter the faint feeling occurring after the blood is seen are not always recognizable. Blood from the lungs or from the pharynx or larynx may be swallowed and when the stomach is filled will be vomited often showing the darker color clots and more or less evidences of digestion seen in bleeding from the stomach. A faint feeling or symptoms of shock may thus precede the external appearance of bleeding. As both types of bleeding are benefited by the same treatment (see p 271) no harm will be done if such treatment is promptly instituted.

### COLON

The colon if much distended especially at the flexures may cause enough upward pressure against the diaphragm to cause some respiratory embarrassment.

### GALLBLADDER AND PANCREAS

Gallbladder and pancreatic pains are often prominent in the chest and have therefore been mistaken for cardiac pain and for the pains of pleurisy or when accompanied by inflammation and leukocytosis for pneumonia.

### LIVER AND BILIARY TRACT

Liver and biliary tract abscesses or neoplasms may press upon or involve or even penetrate the diaphragm causing symptom of intrathoracic disease such as pleurisy with effusion or empyema.

### Respiratory Tract Disease

Symptoms and lesions of the gastrointestinal tract may be simulated or caused by the following respiratory tract diseases.

**INFECTIONS OF THE TONSILS AND SINUSES**

Such infections have long been known as important etiological factors in peptic ulcer and other gastrointestinal diseases. Acute tonsillitis is a frequent forerunner of acute appendicitis. Very large tonsils, excessive postnasal drip, laryngitis and tracheitis may induce nausea and vomiting. Frequent expectoration may induce aerophagia and regurgitation with heartburn.

**LUNG ABSCESS**

Lung abscess with its fever and constant irritating and malodorous cough usually causes constant nausea and often vomiting.

**PNEUMONIA**

Pneumonia, right sided and central, may cause pain and tenderness in the right upper quadrant, fever and leukocytosis. Occasionally such patients are mistakenly operated upon for acute cholecystitis with dire results.

**PLEURISY**

Pleurisy may cause pain easily confused with gallbladder or upper gastrointestinal pain. Adhesions following pleurisy may involve the esophagus and cause dysphagia. Pleural effusion or pneumothorax pushes the heart and esophagus to the opposite side; atelectasis draws them to the affected side.

**HEMORRHAGE**

Hemorrhage from the tonsillar fossa, pharynx, larynx, trachea, bronchi or lungs has been discussed.

**Treatment**

A patient with both gastrointestinal and chronic respiratory disease requires careful attention to both. Infected tonsils should be removed. Treatment should be instituted for diseased sinuses and for bronchial and pneumonic symptoms. In the overall treatment of the patient, adequate diet with added vitamins and minerals is most important.

**Summary**

When definite respiratory diseases are associated with gastrointestinal diseases or bear a causal relationship to them, it is easy to see how confusion can arise in making a diagnosis. It is therefore important in any patient with gastrointestinal symptoms to make a careful and often repeated examination of the respiratory tract together with x rays of the



chest at times with an opaque medium injected into the bronchi as well as sputum examinations cultures and other diagnostic measure

## Genitourinary Diseases and Gastrointestinal Diseases

The close anatomical relations of these organs to the different parts of the gastrointestinal tract account for the frequent association of the symptoms and diseases of these tracts. It is well to consider them in two parts

### The Genital Tract

The genital organs of the *female* are normally in contact with the ileum cecum and appendix on the right side the small intestine and often the transverse colon in the middle and the sigmoid and rectum on the left side. The *male* organs the prostate seminal vesicles and vas deferens do not come in as close association with so many organs except for their intimate relationship to the rectum. It is therefore to be expected that infections may be transmitted from an organ of one tract to the other by direct contact or by the common blood and lymphatic supply. Symptoms may be confused by the common nerve supply. Enlargement of the pelvic organs may cause pressure symptoms and effects as far up as the stomach and liver.

### Pelvic Conditions in Women

Such conditions have been found to be the cause of up to 25 per cent of gastrointestinal symptoms for which patients seek medical care. These symptoms may be listed as follows:

1 *Retrostaltic* gastrointestinal symptoms due to irritation from any pelvic disease including displacements inflammation or neoplasms

2 *Pressure symptoms* on the rectum from displacements especially retroversion cause at times constipation at other times diarrhea from irritation with a frequent desire to defecate

3 Symptoms due to *extensions of pelvic disease* may be caused by inflammation extending from infected pelvic organs to the appendix cecum or sigmoid or by cancer involving contiguous parts of the gastrointestinal tract

4 *Pains in the pelvis* are often interpreted as due to rectal or colonic

disease or appendicitis because of the common nerve supply. There also may be spasms or irritability in the intestinal tract.

5 Symptoms of *endocrine disturbances*. The emotional disturbances occurring at the menstrual period during pregnancy and with the menopause are often reflected in gastrointestinal symptoms varying from anorexia, nausea and vomiting to diarrheas.

6 The *vomiting of pregnancy* in the early months is undoubtedly retrostaltic in origin and can usually be effectively controlled by treatment for this. The later "toxemias" with more persistent vomiting may also usually be controlled by forced feedings and careful attention to the toxemia. In the late months pressure from the large tumor of pregnancy actually encroaches on the stomach and intestines with attendant symptoms.

7 Symptoms of *fistulas* between the vagina and bladder or sigmoid due to inflammatory disease, to sloughing of cancer or to radiologic treatments for cancer include passage of urine or feces per vaginam with usually severe general and retrostaltic symptoms.

### Pelvic Conditions in the Male

In the male pelvis prostatic infections and enlargements, benign or malignant, may cause no gastrointestinal symptoms or may cause irritation, some encroachment on the lumen or actual involvement of the rectum with constipation or diarrhea or only a frequent urge to defecate. To a lesser degree, inflammations of the epididymis, vas deferens and urethra may cause similar irritations. Any of these genital conditions may cause pain or more or less violent retrostaltic symptoms.

### Gastrointestinal Conditions Affecting the Pelvic Organs

Pelvic symptoms can rarely be traced to gastrointestinal diseases per se, although of course rectal diseases and neoplasms will cause pressure on the pelvic organs, involvement with cancer and at times fistulas from the colon or rectum into the vagina. Symptoms of acute appendicitis are occasionally interpreted as due to pelvic inflammatory disease.

### The Urinary Tract

Involvement of the urinary tract in infections or neoplasms of the gastrointestinal tract by contiguity causes confusion between symptoms of both tracts.

In *ileus* the marked distention with increased intra abdominal pressure may cause sufficient pressure or tension on the ureters to obstruct them and add urinary symptoms to the gastrointestinal manifestations.

An *inflamed appendix* may become adherent to the right ureter and after subsidence of the acute process may leave the ureter partly or even completely obstructed with urinary tract symptoms predominating.

The *Urinary Tract* lies in close contact with the gastrointestinal

chest at times with an opaque medium injected into the bronchi as well as sputum examinations cultures and other diagnostic measures

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3 Symptoms due to *extensions of pelvic disease* may be caused by inflammation extending from infected pelvic organs to the appendix, cecum or sigmoid, or by cancer involving contiguous parts of the gastrointestinal tract.

4 *Pains in the pelvis* are often interpreted as due to rectal or colonic

is not sufficient to rule out disease of the urogenital tract. Normal findings or urinalysis may be present in urogenital disease.

Symptoms suggesting such disease and calling for a complete study are as follows:

*Pelvic Symptoms in the Female* Vaginal discharge abnormal or irregular bleeding, dysmenorrhea, dyspareunia, occurrence of symptoms at menstrual or ovulation periods or at the menopause, low backaches or the finding of a large number of epithelial and pus cells in the urine necessitate careful pelvic examination by finger and speculum examination of smears and other examinations by an expert gynecologist.

*Pelvic Symptoms in the Male* Pressure in the rectum increased by defecation or coitus, frequent micturition of small amounts, especially at night, constipation, even with symptoms of obstruction of unknown origin, call for study by a competent urologist. Prostatic expression smears and cultures, cystoscopy, ureteral catheterization and often pyelography are indicated.

*Urinary Tract Symptoms* A history or a finding of pus or blood in the urine, of a tumor in the abdomen, especially on one side, of attacks of colic, especially on one side in the abdomen or kidney region and transmitted downward toward the genitals, of pain in the loins aggravated on standing or of indefinite bowel symptoms and tenderness upon vigorous percussion posteriorly (Murphy's sign) should be considered an indication for adequate urologic study. This would include cystoscopy, pyelography, not only by the intravenous but also by the retrograde method, and various chemical and bacteriological studies. X-ray films taken forty-eight hours after a barium meal when a "pathological appendix" can often be seen filled with barium, combined with ureterograms, will often show an appendix adherent to a ureter at a point of narrowing (Fig. 74 b, p. 410).

### Summary

It is evident that unless detailed history taking and careful examination supplemented by adequate genitourinary studies are carried out, many patients with gastrointestinal symptoms or actual gastrointestinal lesions caused by genitourinary disease will be overlooked and will fail to get well under adequate gastrointestinal care. Many patients have been subjected to exploratory operations when nothing definite was found and to useless operative procedures because no previous genitourinary study had been carried out.

tract On the right side the kidney and ureter lie close to the liver and gallbladder duodenum hepatic flexure ascending colon and cecum on the left side to the stomach spleen splenic flexure descending colon and sigmoid The bladder comes in contact with the rectum and other pelvic organs and with the cecum sigmoid ileum and even transverse colon when distended It is therefore not surprising that half of all urinary tract diseases produce gastrointestinal symptoms and are often overlooked because the patient may show some real gastrointestinal lesion which may or may not have been caused by the urinary tract disease

The diseases of the urinary tract causing gastrointestinal symptoms or actual diseases may cause retrostaltic symptoms and reflexly may cause pains resembling pain in any part of the gastrointestinal tract They include the following

*The Kidneys* Kidney diseases such as infections calculi cysts or neoplasms will cause pains often difficult to distinguish from those caused by liver disease neoplasm or abscess or by gallbladder calculi or other gallbladder disease A palpable kidney whether due to ptosis to enlargement or to perinephric abscess may be confused with liver or gallbladder enlargement with colonic neoplasm or psoas abscess A markedly ptotic kidney lying in the pelvis may be mistaken for a pelvic tumor

Diseases affecting renal function are frequently associated with nausea vomiting and diarrhea when the blood contains an excess of urea and other toxic elements

*The Ureters* The passage of calculi through the ureters or their delay at some point will occasion colicky pains which may be confused with biliary colic appendicitis or colonic spasm The same may be true of ureteral kinking due to nephroptosis The ureter may become adherent to an inflamed tube or ovary which may produce symptoms of ureteral obstruction in addition to the symptoms of adnexal disease and may simulate acute appendicitis

During any abdominal operation a ureter may be damaged or cut across which may occasionally result in a fistula from the renal pelvis or ureter to the duodenum or colon producing diarrhea with urine found in the rectal discharges

*The Bladder* An enlarged bladder distended with urine causing intense cramplike abdominal pain and constipation is often not recognized while a patient is being treated for bowel retention or an abdominal tumor Neoplasms of the bladder also may be confused with rectal or colonic tumors

### Diagnosis

In addition to a complete gastrointestinal study patients who present even mild genitourinary symptoms should be subjected to careful pelvic and urologic studies A simple vaginal or rectal examination or urinalysis

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